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Total Occlusion in Three Coronary Main Vessels, How Could the Patient Survive?: A Case Report

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ABSTRACT

Coronary artery disease (CAD) is one of the most common diseases in developed countries and has a high morbidity and mortality. ST-elevation myocardial infarction (STEMI) accounts for approximately 30% of all acute coronary syndrome (ACS) cases. Total occlusion refers to a complete blockage of a coronary artery, this blockage significantly reduces or completely stops blood flow to the heart muscle. Chronic Total Occlusion (CTO) is defined as a 100% occlusion of a coronary artery that has been present for at least 3 months. CTO can be found in various clinical situations, such as ACS, or discovered during evaluation with imaging modalities when there is a complaint of chest pain, or when the patient undergoes angiography when undergoing valve replacement surgery, or surgery for congenital heart disease. Management of patients with CTO remains a challenge, so more detailed examination is needed to achieve the best results.

Keywords: *Coronary artery disease, Acute coronary syndrome, Chronic total occlusion, Total occlusion*

1. INTRODUCTION

Coronary artery disease (CAD) is one of the most common diseases in developed countries and has high morbidity and mortality [1]. Globally, CHD caused 9 million deaths in 2017 (2). Although the mortality rate among patients with ischemic heart disease has decreased over the past decades, the incidence of acute coronary syndrome (ACS) has not changed and continues to be responsible for billions of health care expenditures worldwide [3]. ACS is a spectrum of conditions that includes patients who experience a change in current clinical condition, have symptoms or signs, with or without changes in 12-lead electrocardiogram (ECG) examination and with or without acute elevation of cardiac troponin values. ACS consists of STEMI and non-ST elevation acute coronary syndrome (NTE-ACS), which is then classified as NSTEMI if there is an increase in cardiac enzyme values, and unstable angina (UA) if there is no myocardial necrosis. Both require immediate action in emergency cases.

The proportion of ST-segment elevation myocardial infarction (STEMI) in total ACS has declined in high-income countries [4], most likely due to lifestyle changes such as declining smoking rates in Western European and North American countries, and partly related to the increasing use of high-sensitivity troponin (hsTn) testing to diagnose non-STEMI (NSTEMI). However, in-hospital mortality rates in patients with complications of STEMI remain high, especially in the setting of heart attacks [5]. In Indonesia itself, based on 2013 data, 0.5% of the Indonesian population was diagnosed with CAD. Furthermore, 1.5% of the population had symptoms of CAD or was diagnosed with CAD [6].

Patient and observation

Patient Information: A 53-year-old man, who works as a construction worker, was brought by his co-worker to the Emergency Room of Dr. Soetomo Hospital with complaints of chest pain as if crushed by a heavy object, VAS (6-7), pain is still felt when resting, persistent, and appears suddenly (new onset), pain accompanied by nausea and vomiting. The patient had felt chest pain for 7 hours before finally being taken to the Emergency Room of Dr. Soetomo Hospital. At the time of examination, the patient was in good conscious condition, complaints of chest pain had decreased (VAS 3-4), complaints of shortness of breath and palpitations were denied. No dyspneu on effort, nocturnal dyspneu, orthopneu, nor swelling in both legs. The patient has smoked 1 pack a day for the past 30 years or so. History of hypertension, Type 2 DM, chronic kidney disease, stroke, previous CAD or congestive heart failure were all denied.

Clinical Findings :

From physical examination, we found normal vital sign with blood pressure 106/72 without support, with pulse 75 times per minute regular, respiratory rate 20 times per minute with oxygen saturation 98% room air. From physical examination of the head and neck no abnormalities were found, from examination of the thorax found symmetrical, and no intercostal retraction. Cardiac auscultation found single S1 S2, regular, no murmur, no gallop. From lung examination found bilateral vesicular breath sounds, no rhales, no wheezing. From examination of the abdomen and extremities found no abnormalities. From upper and lower extremities found warm dry red acral, CRT <2 seconds, no edema in both legs.

From supporting examination, chest x-ray obtained PA projection, less inspiration, normal shape and size impression CTR 48%, aortic calcification, lungs showed no abnormalities (figure 1). From the ECG, sinus rhythm was obtained 64 x/minute, normal frontal axis, horizontal axis CWR, ST Elevation V4-6, QS II III AVF concluded as an acute coronary syndrome anterior and OMI inferior (figure 1). From laboratory examination (Table 1) an increase in Hs Trop I 2731.1 was obtained. From echocardiography found according to the criteria for coronary heart disease with ischaemic cardiomyopathy. Echocardiography also found a decreased of left ventricular systolic function (LVEF by Biplane 41%) with abnormal grade I left ventricular diastolic dysfunction. Diagnostic coronary angiography was found triple vessel disease with total occlusions medial LAD, distal RCA, proximal-distal LCx. Percutaneous primary coronary angiography with stent insertion was conducted in distal RCA occlusions.

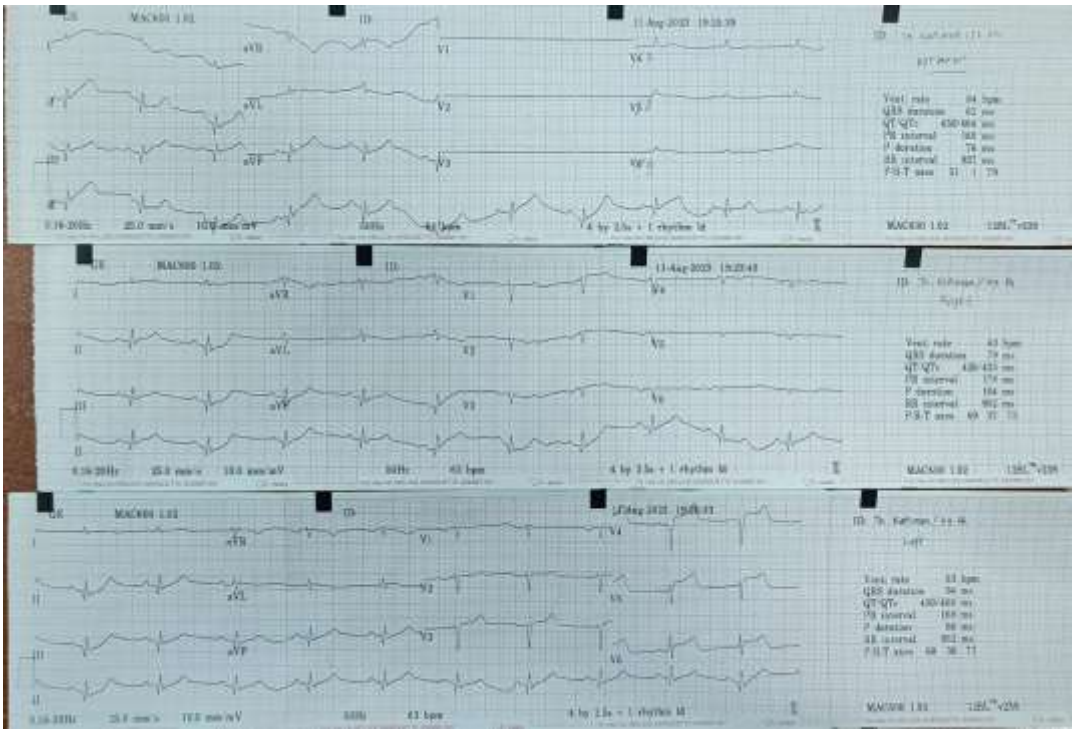

Figure 1. ECG shows normal sinus rhythm 64x /m, normal frontal axis, CWR at horizontal axis, ST elevation at V4-V6, QS II, III, AVF. Seen as acute coronary syndrome anterior, OMI inferior.

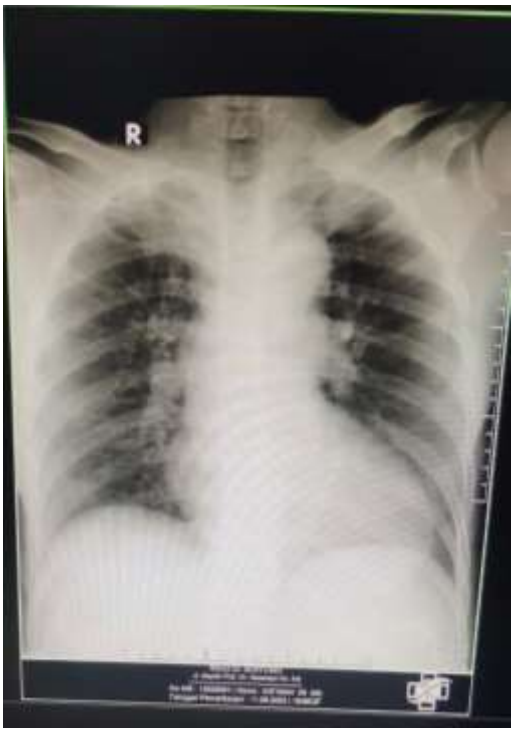

Figure 2. CXR with PA Projection, lack of inspiration, normal of cardiac shape and size, with CTR 48%, calcification of aortic knob, there is no visible nodal and infiltrate at lungs.

BGA	RSDS	
PH	7.36	
PCO2	41	
PO2	74	
HCO3	23.2	
BE	-2.2	
SO2	94%	
P/F Ratio	352	

LABORATORIUM	RSDS
HB	13.8
WBC	11.65
PLT	268
BUN / SK	21.2 / 1.0
Na / K / Cl	142 / 4.6 / 111
Ca/ Mg	9.5 / 2.2
SGOT / SGPT	72 / 32
Albumin	4.40
GDA/ HBA1C	160 / -
INR	1.05
APTT/PPT	31.5 / 14.9
P PCT	0.29
Hs Trop I	2731.1
LDL/ TG	173 / 73
Chol/ HDL	

Table 1. Laboratory results

Timeline of current episode:

After admission to our hospital, we immediately arrange for primary percutaneous coronary angiography (PCI). We give dual antiplatelet (DAPT) loading with aspirin 300mg and ticagelor 180mg as pre-medication. From PCI we found triple critical total occlusion in three main coronary artery. From LAD we found diffuse disease from proximal to distal LAD with total occlusion in distal LAD (Fig 3a blue arrow). From LCx we found the artery were small vessel, with critical total occlusion in proximal LCx (Fig 3b red arrow). However, bridging collateral were found from proximal to distal LCx. Total occlusion was also found in CTA (Fig 3c red arrow).

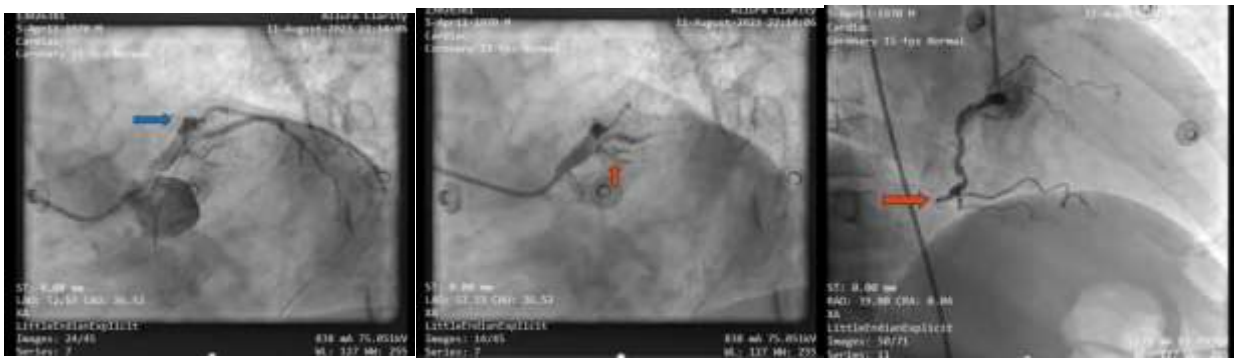

Figure 3. A diffuse disease from proximal to distal LAD with total occlusion in distal LAD (blue arrow). B. From LCx we found the artery were small vessel, with critical total occlusion in proximal LCx (Fig 3b red arrow). However, bridging collateral were found from proximal to distal LCx. Total occlusion was also found in CTA (Fig 3c red arrow).

Diagnostic assessment:

Based on chief complaint of chest pain within 7 hours before admission, ST-T Eleveation ST Elevation V4-6, QS II III AVF from ECG, an increase in Hs-troponin level, echocardiography associated with ischaemic cardiomyopathy in addition with decrease LVEF by Biplane 41% and abnormal grade I left ventricular diastolic dysfunction, and lastly evidence of total occlusion from diagnostic coronary angiography, the patient were diagnosed with STEMI anterolateral-inferior 7 hours onset with triple vessel disease.

Diagnosis: The patient was diagnosed as STEMI anterolateral-inferior 7 hours onset with triple vessel disease (total occlusion in Medial LAD, Distal RCA, Proximal-Distal LCx)

Therapeutic interventions: PCI stenting was conducted to RCA and LAD, by inserting wire DES Resolute (zotarolimus) 3.5 x18 mm guided to distal RCA, with dilatation of 12/10. Other wire was inserted DES Promus (Everolimus) 3.0 x 20 mm to distal LAD, with dilatation of 11/12.

Follow-up and outcome of interventions:

After undergoing Primary Coronary Intervention (PCI), the patient demonstrated a good clinical response with stable hemodynamics and no signs of acute heart failure. Post-procedural electrocardiographic evaluation showed improvement in ST-segment elevation compared to pre-procedure findings, with no significant arrhythmias observed. Post-PCI angiography confirmed successful intervention with TIMI Flow III achieved in the mid-Left Anterior Descending (LAD) and distal Right Coronary Artery (RCA) following stent placement, while stenosis in the Left Main (LM) remained non-significant and Chronic Total Occlusion (CTO) in the Left Circumflex (LCx) persisted with bridging collateral circulation. Clinically, the patient did not experience recurrent chest pain and will undergo serial monitoring of left ventricular function to assess potential residual dysfunction following STEMI. The patient has been educated on lifestyle modifications and adherence to pharmacological therapy to prevent recurrent cardiovascular events. The treatment plan includes optimal medical therapy with dual antiplatelet therapy, high-dose statins, beta-blockers, and risk factor control, along with follow-up echocardiographic evaluation and cardiac rehabilitation to monitor clinical progress. Overall, the intervention successfully improved coronary blood flow, and the patient remains stable post-procedure.

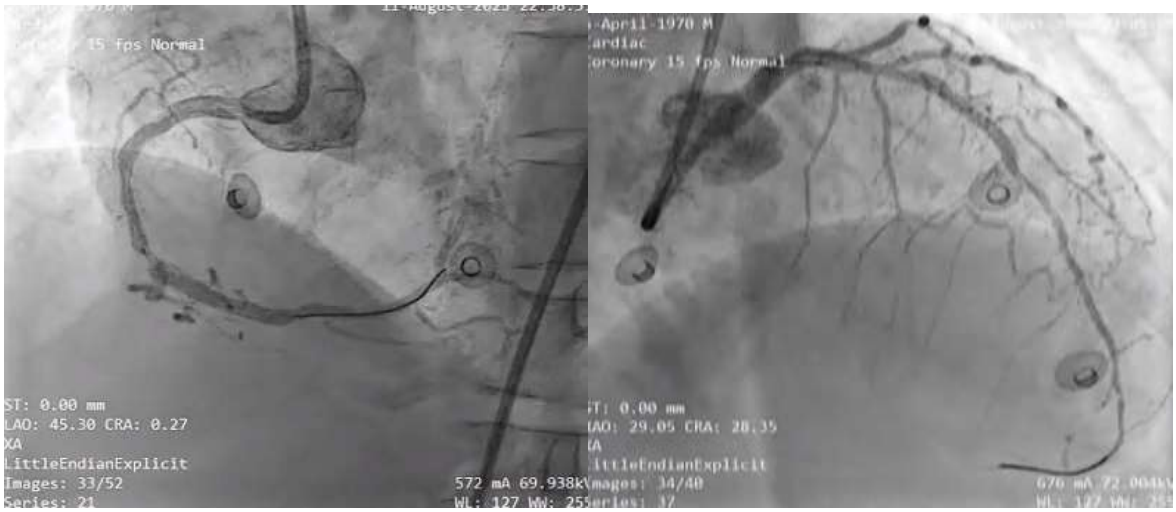

Figure 4. Post Primary Coronary Intervention

Patient Perspective:

It started like any other workday. I was lifting heavy materials, moving back and forth under the sun. I've been doing this for years, so I didn't think much of the exhaustion that crept in. But then, a sharp, crushing pain hit my chest—like a weight pressing down on me. I tried to shake it off, thinking maybe I had just overworked myself. But the pain didn't go away. It stayed, deep and unrelenting, making it hard to focus.I sat down for a while, hoping it would pass. Instead, nausea set in, and I felt like I was going to vomit. My hands were shaking. I didn't want to make a scene, but my coworkers noticed something was wrong. They insisted on taking me to the hospital. I told them it was probably nothing serious, but deep down, I was scared. I had never felt anything like this before. By the time we reached the emergency room, the pain had lessened a bit, but I still felt uneasy. The doctors asked me questions, checked my vitals, and did some tests. I could tell they were looking for something specific. Then they mentioned my heart. That caught me off guard. I've never had any heart problems before—at least, none that I knew of. I don't have high blood pressure, diabetes, or any of those illnesses I always heard about. But I do smoke. I've been smoking a pack a day for 30 years.

When the doctors showed me my test results, my heart sank. They told me I had Acute Coronary Syndrome (ACS), a condition caused by reduced blood flow to the heart. My EKG showed abnormality in some areas, which they said could mean a heart attack, and my troponin levels were very high, confirming that my heart muscle was under stress. Hearing the word "heart attack" made my stomach drop. I never thought this would happen to me. I always saw myself as strong, someone who could push through anything.

Informed Consent: The patient has provided consent for the publication of this case report.

2. DISCUSSION

ST elevation myocardial infarction (STEMI) is a type of heart attack that occurs when a coronary blood vessel is completely blocked. If there is elevation in the ST segment, this often means there is a complete blockage of one of the heart's main supply arteries. If this happens during a heart attack, it can be a sign that the ventricular muscle is dying. This is important information for healthcare providers to know during a STEMI as it means the heart muscle is in the process of dying. It also means reopening those arteries and restoring blood flow as soon as possible can prevent permanent damage, or at least limit the severity of the damage [7].

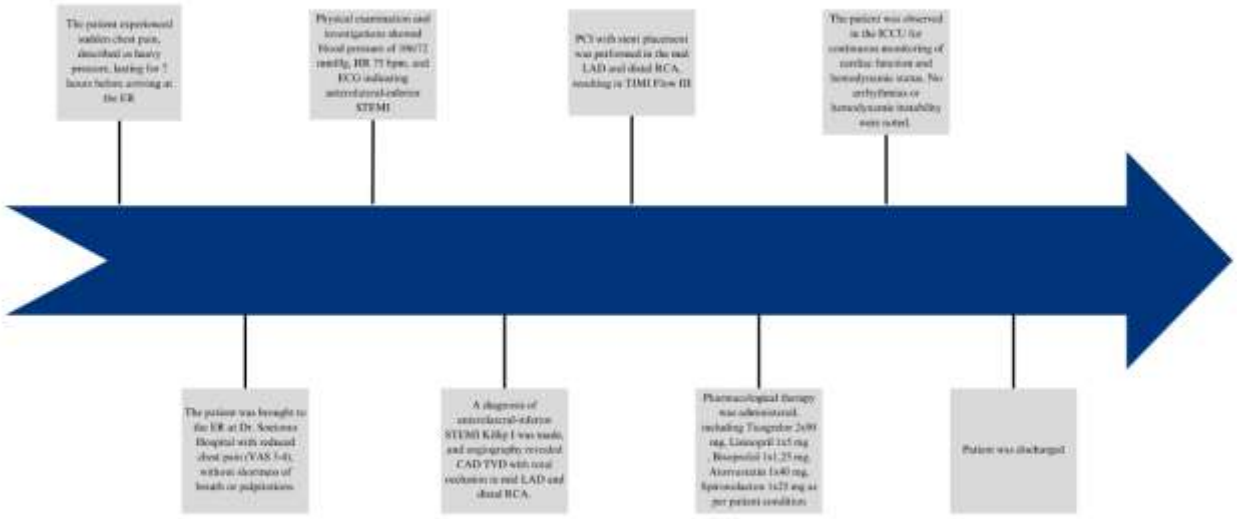

Figure 5. The Case Timeline

Patients that has STEMI experience symptoms such as chest pain that radiates to the shoulders, jaw, neck, arms, or abdomen (solar plexus), may be accompanied by nausea and vomiting, fatigue, and shortness of breath. Risk factors that can cause STEMI are divided into two groups, namely modifiable risk factors such as hypertension, hyperlipidemia, diabetes mellitus, smoking, physical inactivity, and consumption of high salt and high fat foods [8]. While factors that cannot be changed such as age, gender (men are more prone to STEMI than women, and women who have menopause are at higher risk of STEMI than women who are still menstruating, family history of heart disease, and genetic conditions or congenital diseases).

In the US, the incidence of STEMI accounts for approximately 30% of the total incidence of SCI [9]. In Indonesia based on data from the One ACS Registry for 12 months from July 01, 2018 to June 30, 2019 in 11 centers, 7634 cases of SCA were recorded, where STEMI alone was recorded as many as 3725 (48.8%), NSTEMI 1839 (24.1%), and UA as many as 2070 (27.1%). Of these STEMIs, 2428 (65.2%) underwent reperfusion, with 706 (29.1%) undergoing fibrinolysis, and 1703 (70.1%) PPCI [10].

The majority of culprit lesions were located in the left anterior descending coronary artery (LAD, 45%), followed by the right coronary artery (RCA, 38%), and left circumflex coronary artery (LCx, 14%). Subanalysis showed that patients with culprit lesions in the LAD and LCX had significantly higher peak values of cardiac enzymes compared to patients with culprit lesions in the RCA. In addition, patients with proximal LAD and LCX lesions had significantly worse left ventricular function compared to patients with mid or distal lesions [25]. Plaque rupture, which is an important event in SCA, does not occur randomly throughout the coronary arteries. The LAD and right coronary artery RCA are more prone to plaque rupture than the left circumflex artery LCX [26]. This is due to some factors such as location and plague characteristic. Plaque ruptures tend to cluster at specific locations. In the LAD, most ruptures occur in the first 40 mm from the coronary ostium [27]. RCA ruptures are found in both proximal (10-40mm from the ostium) and distal regions [28]. In contrast, LCX ruptures appear more evenly distributed along the coronary artery [28]. Plaque rupture occurs when intraplastic stress exceeds the material strength of the fibrous cover [29]. Factors that determine plaque structural stress (PSS) include luminal area, luminal eccentricity and plaque load. Higher stress in high-risk areas makes plaque rupture more likely [29]. Positive remodeling, while preserving the arterial lumen, may also make atherosclerotic plaques more prone to rupture [29]. Although fibroatheromas (FAs), especially thin cap fibroatheromas (TCFAs), are known to be rupture-prone lesions, there are other factors at play [26]. Plaque Erosion: Although plaque rupture is the main cause of ACS, plaque erosion also contributes [27]. Erosion is more common in the LAD and is associated with factors such as age below 50 years, smoking history, and lack of other coronary risk factors [27]. Other risk factor is flow dynamics. Blood flow dynamics within the coronary arteries may play a role, as plaque erosion is more common near branching [27]. In conclusion, the increased risk of plaque rupture in the LAD and RCA is related to a combination of factors including specific locations prone to rupture, higher plaque structural stress, plaque composition, plaque erosion, and positive remodeling.

In acute coronary syndrome (ACS), total occlusion (TO) refers to a complete blockage of a coronary artery, this blockage acutely and significantly reduces or stops blood flow to the heart muscle, while in CTO a 100% occlusion of a coronary artery has occurred for at least 3 months. The estimated duration of occlusion is based on first-onset classic angina symptoms (or angina equivalents) and/or a history of myocardial infarction (MI) in the target vessel territory. Occluded coronary arteries found within 30 days after MI were not considered as CTOs [31]. From angiography, there are some differences between the two that we can notice such as in TO, non-calcified plaque is often found, plaque density tends to be lower in TO, while in CTO there is diffuse occlusion and calcified plaque. The lesion length in TO was shorter than that in CTO. In CTO, collateral circulation has also developed [32]. The treatment strategy in TO requires immediate reperfusion measures, whereas in CTO the measures can be more conservative, and PCI can be performed to improve the patient's complaints and quality of life (33). In other imaging examinations such as CT scans, the difference between TO and CTO can also be seen from :

- Plaque Density: TOs tend to have lower plaque density measured in Hounsfield units (HU) compared to CTOs.
- Plaque Composition: ATO is often associated with non-calcified plaques, while CTO often shows multifocal calcified plaques.
- Remodeling Index (RI): ATO has a much higher remodeling index (RI) compared to CTO.
- Attenuation Decrease: The percentage of attenuation decrease in the lesion was lower in ATO compared to CTO.
- Myocardial Depletion: Myocardial depletion is only observed in cases of CTO [33].
- Collateral Circulation: CTOs tend to have well-developed collateral circulation [34].

Factors Influencing Survival and Mortality in Total Coronary Occlusion

When a patient has a heart attack with complete occlusion of the three major coronary vessels, several factors may affect his or her survival despite the severity of the condition. Here are some factors that contribute to survival and their effect on mortality in acute coronary syndrome (ACS).

1. Collateral circulation: The presence of collateral vessels may provide an alternative pathway for blood flow to the heart muscle, which may help maintain myocardial viability even when the main coronary arteries are blocked.

2. Timely Medical Intervention: Rapid access to medical treatment, including administration of antiplatelet agents, thrombolytics, or percutaneous coronary intervention (PCI), can significantly improve outcomes. Early reperfusion therapy is critical in restoring blood flow and minimizing heart muscle damage.

3. Patient's Baseline Health: Patients with better baseline cardiac function and fewer comorbidities (e.g., diabetes, chronic kidney disease) are more likely to survive the acute event. Younger patients generally have a better prognosis compared to older adults.

4. Management Strategies: Effective management strategies, including the use of statins, beta-blockers, and ACE inhibitors, have been shown to reduce mortality in ACS patients. These medications help manage risk factors and improve overall cardiovascular health.

5. Cardiac Rehabilitation: Participation in a post-event cardiac rehabilitation program can improve long-term outcomes by encouraging lifestyle changes and providing support for cardiovascular health.

Factors that may be predictors of mortality in SCA are:

1. Age: Older age is a significant predictor of mortality in ACS patients. Studies show that patients aged above 65 years have a higher risk of death (adjusted odds ratio [AOR] 2.143) compared to younger individuals.

2. Clinical Presentation: Certain clinical features, such as cardiogenic shock on presentation, are associated with an increased risk of death (AOR 48.700 for cardiac arrest).

3. Hemodynamic Status: Low systolic blood pressure (SBP <90 mmHg) and increased heart rate (>100 beats per minute) are associated with higher mortality (AOR 4.972 for low SBP) [35].

4. Ejection Fraction: A left ventricular ejection fraction (LVEF) of less than 40% is associated with a significantly increased risk of death (hazard ratio [HR] = 2.75) [36].

5. Comorbid Conditions: The presence of comorbidities such as diabetes, chronic kidney disease, and previous cardiovascular events may adversely affect treatment outcomes [36].

6. Adherence to Treatment: Adherence to dual antiplatelet therapy (DAPT) and treatment according to guidelines after hospital discharge has been shown to improve long-term survival rates [37].

Management of Chronic Total Occlusions

One-third of all angiographically significant coronary stenoses are CTOs, yet only 10–15% of these are treated with percutaneous coronary intervention (PCI), with the majority being medically treated or referred for surgery (CABG) [38]. However, CTO reopening has been shown to not only alleviate angina symptoms but also improve left ventricular ejection fraction, reduce the need for coronary bypass surgery (CABG), and improve long-term survival. CTOs can be found in diverse clinical situations, such as SCA, or discovered during evaluation with imaging modalities when chest pain is complained of, or when patients are angiographed when valve replacement surgery or congenital heart disease surgery is planned [39].

In patients with CTO, it is necessary to carefully evaluate the patient's age, frailty, and comorbidities (e.g., significant concomitant valvular heart disease, large aortic aneurysm, non-cardiac functional capacity limitations, ongoing oncologic treatment, or cognitive deficits). These clinical elements should go hand in hand with technical considerations to guide the decision between CTO-PCI or revascularization and, in the later stages of the treatment, between PCI and surgery. Of note, patients with CTO are older, more often have diabetes, and have greater left ventricular ejection fraction (LVEF) impairment compared to patients without CTO [40].

In stable conditions, the indication for revascularization in CTO is when there are complaints of angina and there is silent ischemia that includes the heart muscle that is supplied with blood by the occluded coronary vessels. A length study to examine collateral function and functional reserve in patients with CTO and their response to pharmacologic stress [41]. It was shown that only 7% of patients had a sufficient increase in collateral flow during stress to achieve a normal coronary flow reserve of >2, while an absolute decrease in flow during hyperemia occurred in one third of patients. These results suggest that even in "well-collateralized" CTOs, maximal hyperemic distal flow is functionally insufficient such that recanalization provides additional benefit. Chung MC et al [42] repeated ventriculography six months after PCI in CTO patients and showed a significant improvement in global LVEF (mean 4%), but a higher improvement (8%) in the group of patients without previous MI in the CTO distribution region.

In CTO patients presenting with SCA, the prognosis is poor due to multiple injuries due to disruption of collateral flow from the culprit artery to the CTO area [43]. Approximately 1/3 of patients resuscitated for cardiac arrest have a CTO [44]. If the resulting SCA results in cardiogenic shock, complete revascularization beyond the culprit lesion should be avoided.

3. CONCLUSION

The management of patients with Chronic Total Occlusion (CTO) remains a challenge. Most of these patients suffer from multivessel coronary artery disease, have undergone previous revascularization, and have other complex target lesions that are typically considered unsuitable for percutaneous intervention. Lifestyle modifications and aggressive control of other cardiovascular risk factors (statins, beta-blockers, and ACE inhibitors) are mandatory. Except for the most evident indications, magnetic resonance imaging or myocardial perfusion scanning should be performed to assess the extent and severity of inducible ischemia in the myocardium related to CTO, allowing for patient stratification and guiding therapeutic decision-making.

REFERENCE

1. Sanchis-Gomar, F.; Perez-Quilis, C.; Leischik, R.; Lucia, A. Epidemiology of coronary heart disease and acute coronary syndrome. Ann. Transl. Med. 2016, 4, 256. [CrossRef] [PubMed]
2. Roth GA, Abate D, Abate KH, Abay SM, Abbafati C, Abbasi N, et al. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the global burden of disease study 2017. Lancet. (2018) 392:1736–88. doi: 10.1016/S0140-6736(18)32203-7
3. Kolansky, D.M. Acute coronary syndromes: Morbidity, mortality, and pharmacoeconomic burden. Am. J. Manag. Care 2009, 15, S36–S41. [PubMed]
4. Collet JP, Thiele H, Barbato E, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J 2021; 42: 1289–367.
5. Omer MA, Tyler JM, Henry TD, et al. Clinical characteristics and outcomes of STEMI patients with cardiogenic shock and cardiac arrest. JACC Cardiovasc Interv 2020; 13: 1211–19.
6. Balitbang Kemenkes RI. (2013). Riset Kesehatan Dasar 2013. https://pusdatin.kemkes.go.id/ resources/download/general/Hasil%20 Riskesdas%202013.pdf. p. 83–99. Accessed 5 June 2022.
7. Cleveland Clinic. 2021. https://my.clevelandclinic.org/health/diseases/22068-stemi-heart-attack
8. Hina Akbar, et al. 2024 Acute ST-Segment Elevation Myocardial Infarction (STEMI). https://www.ncbi.nlm.nih.gov/books/NBK532281/#:~:text=An%20acute%20ST%2Dsegment%20elevation,may%20also%20lead%20to%20STEMI.
9. Martin SS, Aday AW, Almarzooq ZI, et al. 2024 heart disease and stroke statistics: A report of US and global data from the American Heart Association. Circulation 2024;149:e347-e913.
10. Ismir Fahri. 2021 STEMI IN INDONESIA: Social-Economics & Demographic Challenges. Indonesian Heart

Association/ Indonesian Society of Interventional Cardiology Indonesia.https://www.summitmd.com/pdf/pdf/8625_IsmirFahri.pdf
11. https://www.hopkinsmedicine.org/health/conditions-and-diseases/anatomy-and-function-of-the-coronary-arteries
12. https://www.physio-pedia.com/Coronary_Artery
13. Robin Smithuis and Tineke Willems. 2008 Coronary anatomy and anomalies. Radiology department of the Rijnland Hospital Leiderdorp and the University Medical Centre Groningen, the Netherlands. https://radiologyassistant.nl/cardiovascular/anatomy/coronary-anatomy-and-anomalies
14. Normal and variant coronary arterial and venous anatomy on high-resolution CT angiography. Kini S, Bis KG, Weaver L. AJR Am J Roentgenol. 2007;188:1665–1674. doi: 10.2214/AJR.06.1295. [DOI] [PubMed] [Google Scholar]
15. Low coronary wall shear stress is associated with severe endothelial dysfunction in patients with nonobstructive coronary artery disease. Kumar A, Hung OY, Piccinelli M, et al. http://Epub 2018 Sep 26. JACC Cardiovasc Interv. 2018;11:2072–2080. doi: 10.1016/j.jcin.2018.07.004. [DOI] [PMC free article] [PubMed] [Google Scholar]
16. Plaque rupture in coronary atherosclerosis is associated with increased plaque structural stress. Costopoulos C, Huang Y, Brown AJ, et al. JACC Cardiovasc Imaging. 2017;10:1472–1483. doi: 10.1016/j.jcmg.2017.04.017. [DOI] [PMC free article] [PubMed] [Google Scholar]
17. Endothelial shear stress in the evolution of coronary atherosclerotic plaque and vascular remodelling: current understanding and remaining questions. Wentzel JJ, Chatzizisis YS, Gijsen FJ, Giannoglou GD, Feldman CL, Stone PH. Cardiovasc Res. 2012;96:234–243. doi: 10.1093/cvr/cvs217. [DOI] [PubMed] [Google Scholar]
18. Presence of ramus intermedius increases the risk of atherosclerotic plaque formation at the left main bifurcation's region: a retrospective study from CT coronary angiography in UMMC . Rosani NS, Zamin RM, Zulkafli IS, et al. Int. J. Cardiol. 2021;345:19. [Google Scholar]
19. Coronary computed tomography angiography study on the relationship between the ramus intermedius and atherosclerosis in the bifurcation of the left main coronary artery. Zhang DQ, Xu YF, Dong YP, Yu SJ. BMC Med Imaging. 2023;23:53. doi: 10.1186/s12880-023-01009-2. [DOI] [PMC free article] [PubMed] [Google Scholar]
20. Abstract 16364: presence of ramus intermedius artery is associated with higher atherosclerotic plaque burden in the left main stem in coronary ct angiography. Pourafkari L, Kinninger A, Fazlalizadeh H, et al. https://doi.org/10.1161/circ.148.suppl_1.16364 Circulation. 2023;148:0. [Google Scholar]
21. Acute myocardial infarction with simultaneous thrombosis of multiple coronary arteries. Yim TH, Kim JS, Kim BC, et al. https://doi.org/10.15746/sms.15.006 Soonchunhyang Med Sci. 2015;21:24–27. [Google Scholar]
22. Coronary dominance patterns in the human heart investigated by corrosion casting. Abuchaim DC, Spera CA, Faraco DL, Ribas Filho JM, Malafaia O. Rev Bras Cir Cardiovasc. 2009;24:514–518. doi: 10.1590/s0102-76382009000500013. [DOI] [PubMed] [Google Scholar]
23. Prevalence of anatomical variants and coronary anomalies in 543 consecutive patients studied with 64-slice CT coronary angiography. Cademartiri F, La Grutta L, Malagò R, et al. Eur Radiol. 2008;18:781–791. doi: 10.1007/s00330-007-0821-9. [DOI] [PMC free article] [PubMed] [Google Scholar]
24. Anatomy of the lateral, diagonal and anterosuperior arterial branches of the left ventricle of the human heart. Ortale JR, Filho JM, Paccola AMF, et al. Braz J Cardiovasc Surg. 2005;20:149–158. [Google Scholar]
25. Antoni, M. Louisa, et al. 2011 Distribution of culprit lesions in patients with ST-segment elevation acute myocardial infarction treated with primary percutaneous coronary intervention. Coronary Artery Disease 22(8):p 533-536, December 2011. | DOI: 10.1097/MCA.0b013e32834c7552
26. Charis Costopoulos, et al. 2017 Plaque Rupture in Coronary Atherosclerosis Is Associated With Increased Plaque Structural Stress. JACC Cardiovasc Imaging. 2017 Dec;10(12):1472–1483. doi: 10.1016/j.jcmg.2017.04.017
27. Jiannan Dai, et al. 2018 In vivo predictors of plaque erosion in patients with ST-segment elevation myocardial infarction: a clinical, angiographical, and intravascular optical coherence tomography study. European Heart Journal, Volume 39, Issue 22, 07 June 2018, Pages 2077–2085, https://doi.org/10.1093/eurheartj/ehy101
28. Christine Kyme. 2005 Locations of plaque rupture in coronary arteries. Nat Rev Cardiol 2, 495 (2005). https://doi.org/10.1038/ncpcardio0306
29. Carlotta Onnis, et al. 2024 Whys and Wherefores of Coronary Arterial Positive Remodeling. Arterioscler Thromb Vasc Biol. 2024;44:2416–2427. DOI: 10.1161/ATVBAHA.124.321504
30. Peregrine Green, Paul Frobisher, Steve Ramcharitar. 2016 Optimal angiographic views for invasive coronary angiography: a guide for trainees. Br J Cardiol 2016;23:110–3doi:10.5837/bjc.2016.028
31. Lindsey Cilia, et al. 2024 A non-interventional cardiologist's guide to coronary chronic total occlusions. Front. Cardiovasc. Med., 06 February 2024. Sec. General Cardiovascular Medicine Volume 11 – 2024 https://doi.org/10.3389/fcvm.2024.1350549
32. Tasuku Hasegawa, et al. 2013 Differences in the Clinical and Angiographic Characteristics of Chronic Total Oclusion Lesions in the Three Major Coronary Arteries. Wiley Periodicals, Inc. DOI: 10.111/joic.12085
33. Hyon Joo Kwag. 2012 Differentiation of Acute Total Occlusion of Coronary Artery from Chronic Total Occlusion in Coronary Computed Tomography Angiography. J Korean Soc Radiol. 2012 Aug;67(2):93-99. English. Published online Aug 31, 2012.https://doi.org/10.3348/jksr.2012.67.2.93
34. Calum Creaney, Simon J Walsh. 2020 Antegrade Chronic Total Occlusion Strategies: A Technical Focus for 2020.

https://www.icrjournal.com/articles/antegrade-chronic-total-occlusion-strategies-technical-focus-2020
35. Miftah Pramudyo, et al. 2022 Predictors of In-Hospital Mortality in Patients with Acute Coronary Syndrome in Hasan Sadikin Hospital, Bandung, Indonesia: A Retrospective Cohort Study. Acta Med Indones - Indones J Intern Med, Vol 54, Number 3, July 2022
36. Korinan Fanta, et al. 2021 Management and 30-Day Mortality of Acute Coronary Syndrome in a Resource-Limited Setting: Insight From Ethiopia. A Prospective Cohort Study. Front. Cardiovasc. Med., 17 September 2021. Sec. Coronary Artery Disease.Volume 8 - 2021 | https://doi.org/10.3389/fcvm.2021.707700
37. Chun-Yuan Chu, Tsung-Hsien Lin, Wen-Ter Lai. 2017 The Management and Prognostic Factors of Acute Coronary Syndrome: Evidence from the Taiwan Acute Coronary Syndrome Full Spectrum Registry. Acta Cardiol Sin. 2017 Jul;33(4):329–338. doi: 10.6515/ACS20161205A
38. Chistofferson RD, Lehman KG, Martin GV, Every N, Caldwell JH, Kapadia SR. Effect of chronic total coronary occlusion on treatment strategy. Am J Cardiol 2005;95:1088-1091.
39. Davies A, Fox K, Galassi AR, Banai S, Ylä-Herttuala S, Lüscher TF. Management of refractory angina: an update. Eur Heart J. 2021;42:269-83.
40. Azzalini L, Jolicoeur EM, Pighi M, Millán X, Picard F, Tadros VX, Fortier A, L'Allier PL, Ly HQ. Epidemiology, Management Strategies, and Outcomes of Patients With Chronic Total Coronary Occlusion. Am J Cardiol. 2016;118:1128-35.
41. Werner GS, Surber R, Ferrari M, Fritzenwanger M, Figulla HR.The functional reserve of collaterals supplying long-term chronic total coronary occlusions in patients without prior myocardial infarction. Eur Heart J. 2006;27:2406-12.
42. Chung CM, Nakamura S, Tanaka K, Tanigawa J, Kitano K, Akiyama T, Matoba Y, Katoh O. Effect of recanalization of chronic total occlusions on global and regional left ventricular function in patients with or without previous myocardial infarction. Catheter Cardiovasc Interv. 2003:368-74.
43. Nagalli S, Kikkeri NS, Nasir U, Rai Ahuja K, Ali Waheed T, Bhatia N. Trends and In-Hospital Outcomes of Patients Admitted with ST Elevation Myocardial Infarction and Chronic Total Occlusions: Insights from a National Database. Curr Probl Cardiol. 2023;48:101132.
44. Lemkes JS, Janssens GN, van der Hoeven NW, Jewbali LSD, Dubois EA, Meuwissen MM, Rijpstra TA, Bosker HA, Blans MJ, Bleeker GB, Baak RR, Vlachojannis GJ, Eikemans BJW, van der Harst P, van der Horst ICC, Voskuil M, van der Heijden JJ, Beishuizen A, Stoel M, Camaro C, van der Hoeven H, Henriques JP, Vlaar APJ, Vink MA, van den Bogaard B, Heestermans TACM, de Ruijter W, Delnoij TSR, Crijns HJGM, Jessurun GAJ, Oemrawsingh PV, Gosselink MTM, Plomp K, Magro M, Elbers PWG, Spoormans EM, van de Ven PM, Oudemans-van Straaten HM, van Royen N. Coronary Angiography After Cardiac Arrest Without ST Segment Elevation: One-Year Outcomes of the COACT Randomized Clinical Trial. JAMA Cardiol. 2020;5:1358-65.