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#### Medical and Health Science

# Short Term Outcome in Patients Presenting with Acute Coronary Syndrome with Left Main Coronary Artery Disease

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#### **Abstract**:

coronary syndrome (ACS) is a leading cause of mortality and is linked to negative Acute outcomes. Patients with acute coronary syndrome (ACS) who also have left main (LM) disease may have a higher risk of complications and death. Despite considerable improvements in treatment methods, a small number of Patients still suffer from severe hemodynamic compromise and lethal arrhythmia as a result of left main ACS (LMACS). The obstructive left main (LM) coronary artery disease (occurs in about 6% of ACS Patients who undergo coronary angiography) supplies 75-100% of the left ventricular myocardium. Consequently, significant LM stenosis might lead to potentially fatal consequences. In Patients presenting with ST-elevation myocardial infarction (STEMI) requiring rapid revascularization (class IIa recommendation) or unstable angina/non -ST segment elevation myocardial in farction (UA/NSTEMI) who are not candidates for cor onary artery bypass grafting (CABG), current guidelines suggest percutaneous cor onary in tervention (PCI) for LM disease. Multivessel coronary artery disease is the most common cause of LM stenosis; yet, only 6-9% of individuals experience it as an isolated lesion. We want to assess the demographics, symptoms, and prognosis of Patients with ACS who receive LM-PCI in this concise study.

**Keywords:** Acute Myocardial Infarction, Left Main, PCI, Revascularization

#### **Introduction**:

The Wor ld Health Or ganization reports that around 16% of all fatalities in 2019 were caused by Ischemic Heart Disease (IHD), makin g it the to p cause of mor tality that year. sudden cor onary syndromes (ACS) in clude a wide range of disor ders, in cludin g in dividuals who have recently had changes in their clin ical sympto ms or signs, changes on their 12-lead ECG, and sudden in creases in cardiac troponin (cTn) concentrations, whether or not these alterations are evident [1]. Cardiogenic shock (CS), electrical/hemodynamic stability, in Persistent chest pain, and cardiac arrest the many among clin ical manifestations that may accompany acute cor onary syndromes (ACS). Revascularization with CABG surgery has been considered the gold standard therapy left main for obstructive cor onary artery (LMCA) disease because of significant mor bidity and death rates lin ked to impaired myocardial blood supply[2].

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The Or igin s of Acute Cor onary Syndromes

The breakdown of the fibrous cap, leading to plaque rupture, is thought to be the primary cause of cor onary thrombosis. After this happens, the coagulation pathway is activated, vasoconstriction occurs, and platelets clump to gether [3].

For mation of plaque.

Several signals, in cludin g in flammato ry reactions to in sults like bacterial to xin s and more conventional risk facto rs like dyslipidaemia, hypertension, hyperglycaemia, and obesity, can cause blood cells, or monocytes, to adhere to the endothelium of the arterial lumen, leadin g to plaque accumulation at the cellular level. The monocytes clin g to the vascular wall and then move in to the artery in tima, the layer of muscle that is closest to the vessel lumen. Once they undergo this transfor mation, they start in gestin g the modified lipoprotein particles that nor mally collect in the in tima. quick pace; These lipid-filled macrophages, also called foam cells, are characteristic of atherosclerotic plaques and are present in in dividuals with hyperlipidemia. Plaque for mation in side the in tima is a common occurrence when foam cells assemble [4]. The development of plaque

It is mor e probable that physically disruptin g plaques that have become sympto matic is the cause of plaque advancement, rather than a lin ear process [5].

For mation of thrombus

When platelets are activated, their glycoprotein IIb/IIIa recepto rs undergo a change. Thrombus development relies on these recepto rs sin ce they facilitate fibrin ogen's connection, which in turn allows a "mesh" or "aggregation" of platelets to develop and set in motion thrombus for mation [6].

# Algor ithm for ACS

In accor dance with the fourth defin ition of myocardial in farction as outlin ed by the ESC guidelin es In cases of acute myocardial damage accompanied with sympto ms of acute myocardial ischemia, a rise or declin e in cTn values (with at least one value over the 99th

percentile URL) and one of the followin g seven, the diagnosis of acute myocardial in farction should be made:

Ischemia sympto ms, new ischemic ECG changes, pathological Q waves, imagin g evidence of new myocardial viability loss or abnor mal regional wall motion consistent with an ischemic etiology, and the identification of a cor onary thrombus by angiography or auto psy (not for types 2 or 3 MIs) are all criteria for myocardial ischemia.

Type 1 MI criteria are met when acute atherothrombosis is seen in the artery feedin g the in farcted myocardium after death. In dications of an unbalanced supply and demand for myocardial oxygen that is unconnected to atherothrombosis fulfill the requirements for MI type 2. Type 3MI is met when a patient dies from cardiac problems associated with myocardial ischemia and who has suspected new ischemic ECG alterations prior to the availability or abnor mality of cTn values, as shown in table 1 [8].

Persistent ST segment elevation on electrocardiogram (ECG) testin g distin guishes STEMI from non -ST-elevation myocardial in farction (NSTEMI) [9].

Table (1): Classification of UA according to Braunwald (10)

	Clin ical circumstances		
	(A) Develops in presence of extracardiac condition that in tensifies myocardial ischaemia (secondary UA)	(B) Develops in absence of extra- cardiac condition (primary UA)	(C) Develops with in 2 weeks of acute myocardial in farction (post-in farction UA)
	Severity		
I- New onset of severe angin a or accelerated angin a, no rest pain .	IA	IB	IC
II- Angin a at rest with in past month but not with in recedin g 48 h (angin a at rest, subacute )	IIA	IIB	IIC
III- Angin a at rest with in 48 h (angin a at rest, acute )	IIIA	IIIB-Tneg IIIB-Tpos	IIIC

#### Health assessment

## I. The patient's health status:

Acidosis may manifest in a variety of ways in the clin ic. Cardiogenic shock (CS) may be caused by mechanical difficulties such severe mitral regurgitation or Persistent ischemia, or it can be caused by cardiac arrest, electrical in stability, or hemodynamic in stability; nevertheless, some in dividuals may be pain -free when they present [11].

The risk of non -ST-elevation acute cor onary syndrome (NSTE-ACS) in creases with age, male gender, diabetes, hyperlipidemia, smokin g, hypertension, renal dysfunction, prior signs of cor onary artery disease (CAD), and peripheral or carotid artery disease [12].

Physical examin ation

Pay close attention to systo lic murmurs in myocardial in farction Patients [13]. These murmurs could be signs of mitral regurgitation or ventricular septal abnormalities.

#### The ECG results

When evaluatin g in dividuals who may have acute cor onary syndrome (ACS), the 12-lead electrocardiogram (ECG) at rest is the gold standard. Although over 30% of Patients with NSTE-ACS may have nor mal electrocardiograms, there are several hallmark abnor malities to look out for , in cludin g as ST-segment depression, transito ry ST-segment elevation, and T-wave alterations [14].

# Chemical in dicato rs

Accor din g to Thygesen K et al. (2019), it is essential to measure a biomarker of cardiomyocyte damage, ideally hs-cTn, in every patient suspected of havin g NSTEACS. When it comes to detectin g damage to cardiomyocytes, cardiac troponin s outperfor m creatin e kin ase (CK), CK-MB, and myoglobin [15].

# An in flammato ry marker

A higher risk of death is associated with elevated C-reactive protein levels found by a high-sensitivity CRP test. Patients with nor mal troponin levels were able to be classified into low-risk and highrisk groups based on CRP levels; this subset had an overall 14-day death rate of just 1.5%. When C-reactive protein levels were high, the death rate for these in dividuals was 5.8%, whereas it was only 0.4% when C-reactive protein levels were nor mal. It should be noted that the CRP cuto ff value in the ACS condition is about five times higher than in the stable CAD situation (>3 mg/L). An additional straightfor ward in dicato r of flammation is the white blood cell count. Patients with UA/NSTEMI had a greater of death and recurrent myocardial in farction (MI) when their counts were raised [16].

# Non in vasive imagin g techniques in ACS

# • Imagin g of the chest:

Chest radiographs are useful for diagnosin g cardiomegaly and other ischemia-related problems in cludin g pulmonary edema. In addition to ACS, it may help rule out other possible causes of symptoms such a thor acic aneurysm, pneumothor ax, or pneumonia [17].

In the context of acute cor onary syndrome (ACS), echocardiography is a crucial to ol. If the diagnosis is uncertain, echocardiograms may be particularly useful in identifyin g regional wall-motion abnor malities [18].

Radionuclide myocardial perfusion imagin g has a great early sensitivity in detectin g AMI that is not seen in other testin g modalities, and it has good diagnostic and prognostic utility in the emergency situation. Excludin g MI, a typical restin g perfusion imagin g examin ation has a negative predictive value of about 99% [19].

The use of computed to mography (CT) cor onary angiography and CT cor onary artery calcium scor in g has made it possible to detect cor onary artery disease (CAD) with out in vasive procedures, allowin g for earlier treatment befor e the arteries become fully blocked. Additionally, it allows for direct viewin g

of plaque in side the cor onary arteries in addition to the arterial lumen [20].

In trusive angiography of the heart

Cardiac catheterization is useful for determin in g the degree of a patient's ailment and for characterizin g the cor onary architecture. It is imperative that Patients who have cardiogenic shock, Persistent chest pain (even after treatment), significant airway narrowin g, or right ventricular (RV) in farction proceed cardiac catheterization [a systo lic blood pressure (SBP) below 90 mmHg with or gan hypoperfusion is referred to as cardiogenic shock] [21].

In tervention in the Left Main

Improvements in stent technology, procedural techniques and refin ement, periprocedural anticoagulation, concomitant antiplatelet agents, cardiovascular medication, and PCI have all contributed to significant therapeutic advancements in the past 20 years for the treatment of obstructive CAD [22].

Assessin g LMCA illness

Because a big por tion of the myocardium isn't gettin g enough blood, most people with severe LMCA illness have sympto ms. Nevertheless, stable people who undergo cor onary angiography might unin tentionally reveal severe LMCA illness. Additional assessment of the hemodynamic import ance of in termediate or in cidental LMCA lesions is necessary when there is no major stenosis or pertin ent clin ical complain ts [23].

By usin g in travascular ultrasonography (IVUS), the LMCA and its daughter branches may have their vessel size and plaque distribution characterized, allowin g for precise cross-sectional min imum lumen area (MLA) measurements. A prospective research that evaluated IVUS found that an MLA of 6 mm2 or mor e is a safe threshold for postponin g LMCA revascularization. In in dividuals with isolated ostial and shaft in termediate **LMCA** stenosis, another research suggested that a reduced MLA of <4.5 mm2 might be a good in dicato r of functional importance [23]. To prevent overestimating parameters like the MLA at the LCX ostium from a LAD pullback, for in stance, and to adequately define the LMCA bifurcation, it is ideal to utilize a dual pullback from the LAD and the LCX (24).

Endovascular treatment for LMCA illness Patients

Accor din g to the 2018 European Guidelin es and randomized controlled trials comparin g CABG and percutaneous cor onary in tervention (PCI) with drugelutin g stents (DESs), PCI is better than CABG for Patients with left main cor onary artery disease (LMCA) when the lesion is ostial, mid shaft, or has a low to in termediate level of anato mical complexity, and there is a synergy between percutaneous cor onary in tervention and cardiac surgery (SYNTAX) scor e less than 33.

While percutaneous cor onary intervention (PCI) is linked to greater rates of revascularization at follow-up but lower rates of stroke, there seems to be statistically significant difference between CABG and DES implantation in terms of overall major adverse cardiac and cerebrovascular event (MACCE) rates (Ahn et al., 2015), In addition, when it composite safety results, comes to transradial PCI seems to be just as well as CABG for ACS Patients who present with LM illness. When dealin g with distal LM lesions, the stentin g approach is a critical facto r that determin es the result. A greater in cidence of in -stent restenosis and target-lesion revascularization was related with both the necessity for numerous stents and the T-stentin g approach [26].

PCI approach and methodology

One clin ically relevant concern in the modern percutaneous cor onary in tervention (PCI) context of left main cor onary artery disease (LMCA) is the wide variation in effectiveness and safety results across modern DES. In a combin ed examin ation of 4,470 Patients with

LMCA illness who had percutaneous cor onary in tervention (PCI) with DES of the second generation, there were no statistically significant differences in the 3-year risk of target-vessel failure across the various DES. Furthermor e, with out exception, no DES had an extraor din arily low rate of defin itive stent thrombosis (<1.0%) [27].

Operato rs with mor e expertise (defin ed as those who have done 15 or mor e LMCA PCIs annually for three years in a row) are related with better shor t- and long-term results when conductin g LMCA PCIs. Compared to PCI of the distal LMCA bifurcation, PCI of the ostium or shaft of the LMCA is an easier technique and is lin ked with a reduced need for recurrent revascularization. The ostium of the LMCA is mor e densely packed with elastic tissue and smooth muscle cells than the rest of the LMCA and its branches. therefor e it's important to pay attention this area to make sure the stent expands enough [28].

Sin ce it is both technically easier and produces results that are comparable to or even better than a systematic 2-stent method, provisional stentin traditionally been recommended for bifurcation lesions. strategy. Up to 60% of LMCA bifurcation procedures have made use of the sin gle-stent crossover approach practice. Two randomised controlled trials (RCTs) evaluatin g techniques for treatin g real distal LMCA bifurcations compared the double-kissin g crush method to culotte and provisional stentin g, castin g doubt on this. The main composite ischemia endpoin substantially decreased usin g the doublekissin g crush approach in both in vestigations [29].

Because of the high in cidence of sidebranch impairment after crossover stentin g, the LCX ostium's disease in volvement should in for m the decision to use a sin gle-or 2-stent method. Thus, in travascular ultrasound (IVUS) accurately measures the disease state in LMCA bifurcation lesions, both in the main and side branches, allowin g for the determin ation of whether a sin gle or two stent approach should be used. Carin a shift, reduced MLA, increased eccentricity of external elastic membrane, and carin a angle between the LAD and the LCX were the primary geometric alterations in the LCX ostium after main -stent crossin g from the proximal LAD to the LM. Prior to proceedin g with further LCX therapy, FFR assessment should be evaluated in situations where the ostium is substantially damaged (>50%) after provisional stentin g [30].

# Differentiatin g Acute Cor onary Syndrome Patients ' Risk s

Acute cor onary syndrome (ACS) Patients 'prognoses have greatly improved durin g the last several decades. Non etheless, attempts to for ecast the prognosis of ACS Patients are ongoin g. Angiographic data, ratin g systems, and many biomarkers all poin t to encouragin g outcomes [31].

Biological markers

While left ventricular systo lic dysfunction is not always present, it has been proposed that transito ry myocardial ischemia might be the source of an in creased N-termin al fraction of brain natriuretic peptide (NTproBNP) level. An improved prognosis (death, onset of left ventricular systo lic dysfunction) is reliably predicted by an in creased NT-proBNP level in **Patients** with ACS. Patients with acute cor onary syndrome (ACS) (Class IIa, level B) should be considered for prognosis based on NT-proBNP, accor din g to recommendations from European Society of Cardiology (ESC) [32].

An elevated troponin level is associated with a higher mor tality risk , and compared to high-sensitivity troponin (hs-Tn)-I, hs-Tn-T seems to be the mor e accurate predicto r. For in dividuals with ACS (Class I, level B), the current ESC recommendations in clude repeated troponin measurements to ascertain

prognosis [32]. In those who do not have cor onary syndrome (ACS), acute troponin is thought to be a risk for death from any cause. Patients with slightly in creased troponin who were the emergency room for brought to suspected ACS but were later released with out an acute cardiovascular event had a higher death rate. To ascertain the mor tality risk in ACS Patients, a panel several biomarkers has suggested. There is an in crease in prognostic value when Patients are stratified based on the number of creased biomarkers [33].

## Assessin g Danger Levels

In their wor k, Califf et al. presented one of the first methods of scor in g. The angin a scor e in cludes a handful of straightfor ward clin ical facto rs that have in dependent predictive value, in cludin g the angin a course, angin a frequency, and ST-T alterations [34]. After that. suggested Braunwald a clin ical presentation-based categor ization for UAP. The groups were further classified based on whether or not UAP developed with in two weeks after myocardial in farction (MI) [35], or whether there were any extra-cardiac facto rs, in cludin g in fection or hypoxia, that might wor sen myocardial ischemia.

Rigid data gathered from 94 hospitals in 14 countries for med the basis of the GRACE research. There are nin e facto rs that contribute to the risk scor e. Upgraded GRACE 2.0 for enhanced discrimin ation and user-friendlin ess. Thirty days after admission. PURSUIT scor e estimates the likelihood of mor tality or death with MI. Low-risk , in termediate-risk , and high-risk categor ies were established for Patients . Patients with STE-ACS who undergo PCI alone are the first to have their prognosis assessed usin g the PAMI scor in g method, which uses a one-year follow-up period. Bein g above the age of 75 is the sin gle most important factor. One potential drawback is the limited data

obtain ed from the catheterization labor ato ry [36].

Risk of Bleedin g and Other Considerations

There is an in creased risk of bleedin g associated with treatment with antiplatelet therapy (DAPT). For the purpose of predictin g the likelihood of bleedin g in DAPT Patients, the PRECISE-DAPT scor e was suggested. The effectiveness of reducin g DAPT duration (<12 months) in higher-risk **Patients** (PRECISE-DAPT >25) was shown in an evaluation of over 15,000 in dividuals. In these in dividuals, there is no anti-ischemic benefit to prolonged DAPT. On the other hand, Patients with a low risk of bleedin g (PRECISE-DAPT <25) could benefit from extendin g DAPT (>12 months) with out in creasin g the risk bleedin g. Hence, if a significant risk bleedin g is present, the PRECISE-DAPT scor e could identify Patients with ACS who are at high risk and would benefit from a shor tened DAPT. Hospital and post-discharge bleedin g risk as well as in -hospital mor tality may be predicted usin g the HAS -BLED scor in g system. A higher scor e in dicates a greater probability of hemor rhage or death while hospitalized [37].

Standard scor in g systems have been the subject of several evaluation effor ts. There may be a value for the CHA2DS and CHA2DS2-VASc scor es in Patients with ACS, despite their primary usage in atrial fibrillation. In Patients with any kin d of ACS, the CHA2DS2-VASc scor e stands alone as a predicto r of future MACE. The chance of major adverse cardiac event (MACE) is 3.8 times higher in the extremely high-risk categor y compared to the low-risk group. There agreement some between CHA2DS2-VASc scor e and other wellestablished risk measures, such as TIMI and GRACE (38).

#### **Conclusions**

Even after a successful percutaneous cor onary in tervention (PCI), this research

found that adverse cardiac events (ACS) caused by severe left ventricular stenosis are associated with a significant death rate. The greatest in dependent predicto rs of in -hospital complications in Patients with acute cor onary syndrome who received primary in tervention to the sick left main artery were abnor mal troponin and CKMB values, as well as stent length. These days, LM-ACS Patients who have had prior revascularization are better of f when it comes to percutaneous cor onary in tervention (PCI).

#### References

- [1] AN Nowbar, M Gitto, JP Howard, DP Francis, R Al-Lamee. Mor tality From Ischemic Heart Disease. Circ Cardiovasc Qual Outcomes. 2019;12(6):e005375.
- [2] RA Byrne, X Rossello, JJ Coughlan, E Barbato, C Berry, A Chieffo, et al. 2023 ESC Guidelin es for the management of acute cor onary syndromes. Eur Heart J. 2023;44(38):3720-3826.
- [3] P Theof ilis, E Oikonomou, C Chas ikidis, K Tsioufis, D To usoulis. Pathophysiology of Acute Cor onary Syndromes-Diagnostic and Treatment Considerations. Life (Basel). 2023;13(7).
- [4] Z Han, Q Liu, H Li, M Zhang, L You, Y Lin, et al. The role of monocytes in thrombotic diseases: a review. Front Cardiovasc Med. 2023;10:1113827.
- [5] A Shioi, Y Ikari. Plaque Calcification Durin g Atherosclerosis Progression and Regression. J Atheroscler Thromb. 2018;25(4):294-303.
- [6] A Scridon. Platelets and Their Role in Hemostasis and Thrombosis-From Physiology to Pathophysiology and Therapeutic Implications. In t J Mol Sci. 2022;23(21).
- [7] P Widimsky. Classification and defin ition of acute cor onary syndromes A time for change? Cor et Vasa. 2014;56(4):e279-e284.

- [8] K Thygesen, JS Alpert, AS Jaffe, BR Chaitman, JJ Bax, DA Mor row, et al. Fourth Universal Defin ition of Myocardial In farction (2018). J Am Coll Cardiol. 2018;72(18):2231-2264.
- [9] B Ibanez, S James, S Agewall, MJ Antunes. C Bucciarelli-Ducci, Bueno, et al. 2017 ESC Guidelin es for the management of acute myocardial in farction in Patients presenting with ST-segment elevation: The Task For ce the management of for acute myocardial in farction in **Patients** presentin g with ST-segment elevation European Society of the Cardiology (ESC). Eur Heart 2018;39(2):119-177.
- [10] CW Hamm, E Braunwald. A classification of unstable angin a revisited. Circulation. 2000;102(1):118-122.
- [11] M Rof fi, C Patrono, JP Collet, C Mueller, M Valgimigli, F Andreotti, et al. 2015 ESC Guidelin es for the management of acute cor onary syndromes in Patients presentin g with out Persistent ST-segment elevation: Task For ce for Management of Acute Cor onary Syndromes in Patients Presentin g with out Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). Eur Heart J. 2016;37(3):267-315.
- [12] M Rubin i Gimenez, M Reiter, R Twerenbold, T Reichlin, K Wildi, Haaf P, et al. Sex-specific chest pain characteristics in the early diagnosis of acute myocardial in farction. JAMA In tern Med. 2014;174(2):241-249.
- [13] B Ibanez, S James, S Agewall, MJ Antunes, C Bucciarelli-Ducci, Bueno H, et al. 2017 ESC Guidelin es for the management of acute myocardial in farction in **Patients** presentin with ST-segment g elevation: The Task For ce for the management of acute myocardial in farction in Patients presentin g with

- ST-segment elevation of the European Society of Cardiology (ESC). European Heart Journal. 2017;39(2):119-177.
- [14] AR Chapman, ASV Shah, KK Lee, A Anand, O Francis, P Adamson, et al. Long-Term Outcomes in Patients With Type 2 Myocardial In farction and Myocardial In jury. Circulation. 2018;137(12):1236-1245.
- [15] K Thygesen, JS Alpert, AS Jaffe, BR Chaitman, JJ Bax, DA Mor row, et al. Fourth universal defin ition of myocardial in farction (2018). European Heart Journal. 2018;40(3):237-269.
- [16] RY Brzezin ski, S Banai, M Katz Shalhav, M Stark, I Goldin er, O Rogowski, et al. The CRP troponin test (CTT) stratifies mor tality risk in Patients with non -ST elevation myocardial in farction (NSTEMI). Clin Cardiol. 2024;47(4):e24256.
- [17] S Amanullah, J Pershad, Chapter 18 Chest Pain . In : RP Olympia, RM O'Neill, ML Silvis, edito rs. Urgent Care Medicin e Secrets: Elsevier; 2018. p. 94-101.
- [18] G Marrazzo, S Palermi, F Pasto re, M Ragni, A Mauriello, A Zambrano, et ST-Elevation Enhancin g Myocardial In farction Diagnosis and Management: The In tegral Role of Echocardiography in **Patients** Rushed Cardiac to the Catheterization Labor ato ry. J Clin Med. 2024:13(5).
- [19] G Pons-Lladó, P Kellman. State-of the-Art of Myocardial Perfusion by CMR: A Practical View. Rev Cardiovasc Med. 2022;23(10):325.
- [20] CO DuBose, K Youngman, D Barymon. Cor onary Computed To mography Angiography and Calcium Scor in g. Radiol Technol. 2019;90(3):259ct-275ct.
- [21] FJ Neumann, M Sousa-Uva, A Ahlsson, F Alfonso, AP Bannin g, Benedetto U, et al. 2018 ESC/EACTS Guidelin es on

- myocardial revascularization. Eur Heart J. 2019;40(2):87-165.
- [22] PH Lee, JM Ahn, M Chang, S Baek, SH Yoon SJ, Kang, et al. Left Main Cor onary Artery Disease: Secular Trends in Patient Characteristics, Treatments, and Outcomes. J Am Coll Cardiol. 2016;68(11):1233-1246.
- [23] SJ Park, SJ Kang, JM Ahn, EB Shim, YT Kim, SC Yun, et al. Visual-functional mismatch between coronary angiography and fractional flow reserve. JACC Cardiovasc In terv. 2012;5(10):1029-1036.
- [24] SC Cho, DW Park, SJ Park. Percutaneous Cor onary In tervention and Cor onary Artery Bypass Graftin g for the Treatment of Left Main Cor onary Artery Disease. Kor ean Circ J. 2019;49(5):369-383.
- [25] Y Ahmad, JP Howard, AD Arnold, CM Cook, M Prasad, ZA Ali, et al. Mor tality after drug-elutin g stents vs. cor onary artery bypass graftin g for left main cor onary artery disease: a meta-analysis of randomized controlled trials. Eur Heart J. 2020;41(34):3228-3235.
- [26] F Gao, YJ Zhou, ZJ Wang, ZX Yan, XL Liu, H Shen. Transradial Cor onary Intervention Versus Cor onary Artery Bypass Graftin g for Unprotected Left Main and/or Multivessel Disease in Patients With Acute Cor onary Syndrome. Angiology. 2016;67(1):83-88.
- [27] PH Lee, O Kwon, JM Ahn, CH Lee, DY Kang, JB Lee, et al. Safety and Effectiveness of Second-Generation Drug-Elutin g Stents in Patients With Left Main Coronary Artery Disease. J Am Coll Cardiol. 2018;71(8):832-841.
- [28] B Xu, B Redfor s, Y Yang, S Qiao, Y Wu, J Chen, et al. Impact of Operato r Experience and Volume on Outcomes After Left Main Cor onary Artery Percutaneous Cor onary In tervention. JACC Cardiovasc In terv. 2016;9(20):2086-2093.

- [29] SL Chen, JJ Zhang, Y Han, J Kan, L Chen, C Qiu, et al. Double Kissin g Crush Versus Provisional Stentin g for Left Main Distal Bifurcation Lesions: DKCRUSH-V Randomized Trial. J Am Coll Cardiol. 2017;70(21):2605-2617.
- [30] V Paradies, A Bannin g, D Cao, A Chieffo, J Daemen, R Diletti, et al. Provisional Strategy for Left Main Stem Bifurcation Disease: A State-of -the-Art Review of Technique and Outcomes. JACC: Cardiovascular In terventions. 2023;16(7):743-758.
- [31] D Bauer, P To ušek. Risk Stratification of Patients with Acute Cor onary Syndrome. J Clin Med. 2021;10(19).
- [32] JP Collet, H Thiele, E Barbato, O Barthélémy, J Bauersachs, DL Bhatt, et al. 2020 ESC Guidelin es for the management of acute cor onary syndromes in Patients presentin g with out Persistent ST-segment elevation. Eur Heart J. 2021;42(14):1289-1367.
- [33] M Maayah, S Grubman, S Allen, Z Ye, DY Park, E Vemmou, et al. Clin ical In terpretation of Serum Troponin in the Era of High-Sensitivity Testin g. Diagnostics. 2024;14(5):503.

- [34] RM Califf, DB Mark, FE Harrell, Jr., MA Hlatky, KL Lee, RA Rosati, et al. Importance of clinical measures of ischemia in the prognosis of Patients with documented coronary artery disease. J Am Coll Cardiol. 1988;11(1):20-26.
- [35] E Braunwald, A Unstable angin. A classification. Circulation. 1989;80(2):410-414.
- [36] EW Tang, CK Wong, P Herbison. Global Registry of Acute Cor onary Events (GRACE) hospital discharge risk scor e accurately predicts long-term mor tality post acute cor onary syndrome. Am Heart J. 2007;153(1):29-35.
- [37] D Castin i, S Persampieri, L Sabatelli, M Erba, G Ferrante, F Valli, et al. Utility of the HAS -BLED scor e for risk stratification of Patients with acute cor onary syndrome. Heart Vessels. 2019;34(10):1621-1630.
- [38] H Peng, Z Sun, H Chen, Y Zhang, X Din g, XQ Zhao, et al. Usefulness of the CHA(2)DS(2)-VASc Scor e to Predict Adverse Outcomes in Acute Cor onary Syndrome Patients With out Atrial Fibrillation Undergoin g Percutaneous Cor onary In tervention. Am J Cardiol. 2019;124(4):476-484.