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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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MAJOR ADVERSE CARDIOVASCULAR EVENTS AFTER ACUTE CORONARY SYNDROME: DEFINITION, TIMING, RATES, MAIN RISK FACTORS, AND WAYS TO IMPROVE

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Abstract.

Aim of study: This review summarizes current evidence on MACE definitions, the timing and magnitude of post-ACS risk, its main drivers, and evidence-based risk-reduction strategies, and identifies key methodological gaps.

Materials and methods: This narrative synthesis includes randomized controlled trials, meta-analyses, large registries, and observational studies reporting MACE after ACS, identified through PubMed and cross-referenced guideline documents. No systematic search protocol or quantitative synthesis was performed; priority was given to peer-reviewed publications, current guidelines, and high-impact meta-analyses, with sources cited for pooled estimates.

Results: MACE definitions are heterogeneous. The classical 3-point composite (cardiovascular death, nonfatal myocardial infarction, nonfatal stroke) is the regulatory standard, but 4- and 5-point versions incorporating revascularization, unstable angina, or heart-failure hospitalization are common, and administrative datasets often substitute all-cause mortality. Broader definitions inflate event rates and can affect interpretation of treatment effects. Data from one of the largest meta-analyses indicate that nearly half of the adverse events during the first year occur within 90 days, with an overall 90-day MACE rate of 4.1%. However, it should be noted that one-year rates range from 3.6% in a small cohort of lower-risk patients to 34.4% in a large national registry, reflecting differences in patient composition, the completeness of revascularization performed, and definitions of MACE components. The strongest predictors of post-ACS MACE were clinical severity (Killip class >II, cardiogenic shock/arrest, LVEF ≤40%), comorbidity burden (chronic kidney disease, diabetes, hypertension, dyslipidemia, smoking, age/frailty), and anatomical/procedural factors (multivessel disease, incomplete revascularization, suboptimal TIMI flow). Complete revascularization, high-intensity statin therapy, individualized dual antiplatelet duration, and structured secondary prevention were each associated with reduced MACE.

Conclusions: Post-ACS MACE clusters within 90 days after discharge. However, reported incidence varies widely due to heterogeneous endpoint definitions and population differences. Standardizing MACE definition and adjudication reporting, and designing trials powered for early endpoints, would meaningfully improve evidence synthesis. Clinicians should prioritize complete revascularization, optimized lipid-lowering, individualized antithrombotic therapy, and early, intensive secondary prevention.

Key words. Acute coronary syndrome, major adverse cardiovascular events (MACE), composite endpoints, timing, risk factors, secondary prevention, narrative review.

Introduction.

Patients' prognosis after acute coronary syndrome (ACS) has improved in recent decades, largely due to advancements in acute-phase treatment and effective secondary prevention efforts. The composite endpoint of MACE (Major Adverse Cardiovascular Events) is most frequently used in cardiovascular research to assess subsequent risk after the index ACS event. In 2008, the United States Food and Drug Administration (FDA), followed by the European Medicines Agency (EMA) in 2012, guided the use of a three-point MACE outcome for cardiovascular safety assessment, helping establish cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke as a widely used composite endpoint [1,2]. However, the available literature shows a lack of a standardized definition of MACE, with variation in the components included across studies, complicating direct comparisons of outcomes.

This narrative review summarizes current evidence on MACE definitions, its occurrence after ACS, associated risk factors, and identifies the key methodological gaps that limit comparison across the existing evidence.

Methods.

A narrative literature search was conducted in PubMed, supplemented by cross-referencing contemporary guideline documents, using combinations of terms related to acute coronary syndrome, MACE, cardiovascular risk factors, revascularization, lipid-lowering therapy, antiplatelet therapy, SGLT2 inhibitors, GLP-1 receptor agonists, anti-inflammatory therapy, and secondary prevention. Randomized controlled trials, meta-analyses, large registries, observational studies, and current guidelines were considered eligible; priority was given to peer-reviewed, contemporary evidence, while older landmark publications were retained when they provided clinically or methodologically important findings. Publications included in the final narrative synthesis ranged from 2007 to 2025. No formal systematic-review protocol or quantitative synthesis was performed, consistent with the narrative design of this review.

MACE composition:

The most common composition of the MACE endpoint varies from study to study, but several common formulations exist:

- Classical 3-Point MACE: This is the most traditional and frequently used definition in major clinical trials, often guided by regulatory bodies like the U.S. Food and Drug Administration (FDA) [2]. It comprises a composite of cardiovascular death, non-fatal myocardial infarction (MI), and non-fatal stroke [2].
- 4-Point MACE: This definition expands upon the 3-point MACE by adding a component, most commonly hospitalization for unstable angina or the need for ischemia-driven coronary revascularization [2].

● 5-Point MACE: This further broadens the endpoint by including hospitalization for heart failure (HF) [2]. The inclusion of HF is significant, as it captures a different dimension of cardiac morbidity related to ventricular dysfunction and remodeling post-MI.

Beyond this common MACE structure, several different MACE definitions are also used. While MACE is now better defined and a more ubiquitous outcome among Randomized Clinical Trials (RCTs), its use in observational studies to assess the safety and real–world effectiveness of therapies remains less clear. First, in comparison to RCTs, observational studies often do not report their protocols, including outcome definitions. Observational studies using administrative databases, where the specific cause of death may be unavailable, often use all-cause mortality instead of cardiovascular death [2]. Other study-specific definitions may include endpoints such as stent thrombosis, repeat revascularization, pericarditis, or even venous thromboembolism, depending on the study endpoint [2]. A systematic review of observational studies using administrative data found that only a small fraction (8.6%) used the traditional 3-point MACE definition, highlighting the extent of this issue [2]. Even when MACE components are well defined, such as Myocardial Infarction (MI) or Stroke, another challenge encountered in observational studies is how to define outcomes using diagnostic codes available from administrative databases. Furthermore, 67% of studies did not validate outcomes or cite validation studies, while 70.7% did not report the code positions used to define endpoints [2].

The choice of MACE composition has a direct, quantifiable impact on study results and their interpretation. In an empirical analysis of 6,922 patients from the DEScover registry, broadening the MACE definition from a safety-only composite (death, myocardial infarction, or stent thrombosis; 362 events, 5.2%) to include target vessel revascularization (674 events, 9.7%) and then any repeat revascularization (868 events, 12.5%) more than doubled the observed 1-year event rate in the same patients over the same follow-up period — a 2.4-fold increase driven entirely by which outcomes were counted, not by any difference in the underlying population [3-13].

Timing of MACE:

The period following discharge after an acute coronary syndrome is characterized by a dynamic and significantly elevated risk of subsequent MACE. Analysis of data from large-scale meta-analyses, clinical registries, and cohort studies reveals an exceptionally high concentration of events occurring in the immediate post-discharge phase, followed by a persistently lower risk over the first year. The reported incidence rates vary from study to study, largely due to heterogeneity in MACE definitions [4].

A large meta-analysis of seven major Phase 3 interventional trials, with data from 82,727 high-risk ACS patients enrolled, provides the most compelling evidence for this early hazard. The analysis found that nearly half—approximately 49%—of all MACE that occurred within the first year transpired within the first 90 days post-event. The pooled rate of recurrent MACE at this 90-day mark was 4.1% (95% Confidence Interval [CI]: 3.0%-5.7%). The duration of follow-up ranged from 12 to 36 months. Various interventions were evaluated, including P2Y12 inhibitors in three studies (TRITON-TIMI 38, PLATO, and TRILOGY ACS), antidiabetic agents in two studies (EXAMINE and AleCardio), a factor Xa inhibitor in one study (ATLAS ACS 2-TIMI 51), and a lipoprotein-associated phospholipase A2 inhibitor in one study (SOLID-TIMI 52). Most patients received standard of care secondary prevention medications, including aspirin (90.6%–99%), P2Y12 inhibitor (79.8%–93.0%), statin (83.1%–94.9%), BB (65.6%–89.7%), and ACEI or ARB (38.2%–88.2%) [4]

Data from smaller cohort studies support the evidence coming from meta-analysis. A study conducted at Hue Central Hospital in Vietnam on 337 post-PCI patients observed that nearly 75% of all MACE recorded over a 12-month follow-up period occurred during the index hospitalization and within the first 30 days after discharge [14,15]

The 12-month MACE rate is a standard endpoint in cardiovascular research, but the reported rates are highly variable. This variance reflects methodological differences between studies, including MACE definition, study design (observational registry vs. randomized controlled trial), and the baseline risk profile of the patient population.

The spectrum of reported 1-year MACE rates includes the lowest rate of just 3.6% for a Vietnamese cohort study in 337 post-PCI patients [15] vs. a large Finnish national registry study involving over 12,000 ACS survivors, which found a 1-year MACE rate of 34.4% [16-18].

The low rate may reflect the small single-center design and selected post-PCI population. In contrast, the high rate reported in the Finnish registry should be interpreted in the context of both the earlier study period (1993–2011) and its broad MACE definition, which included recurrent ACS, heart failure, stroke, other cardiovascular events, and all-cause death. The same registry reported a cumulative MACE incidence of 48.4% at three years, illustrating the persistence of substantial long-term residual risk [18].

Analyzing already available data, it is clear that the risk of MACE after ACS is not linear. It follows a specific trajectory characterized by an early, intense "vulnerable period" followed by a phase of persistent, chronic risk. The first 90 days are dominated by the acute pathophysiological sequelae of the index event—inflammation, thrombosis, and myocardial stunning. This explains why nearly half of all first-year events are concentrated in this window. After this period, the risk profile begins to shift, with events being driven more by the underlying burden of atherosclerosis, the presence of comorbidities, and the effectiveness of long-term secondary prevention. The table below shows the key incidence data from various sources, comparing the rates of MACE, study type performed, and MACE composition used.

This wide range underscores that a single "true" 1-year MACE rate does not exist; rather, the expected rate is highly contingent on the population being studied and the outcomes being measured.

Factors influencing the risk of MACE:

Understanding the incidence and timing of MACE is not enough for effective patient management and prevention. A

patient's prognosis after an ACS is determined by a complex interplay of their baseline clinical characteristics, the severity of the index event itself, and the effectiveness of the therapeutic interventions they receive [8,19,20-27]. A patient's pre-existing health status is very important for future cardiovascular risk. Large-scale meta-analyses and registry studies have identified a core set of comorbidities that independently and significantly increase the likelihood of MACE following an ACS: diabetes mellitus, arterial hypertension, dyslipidemia, chronic kidney disease, and history of smoking. Beyond these traditional risk factors, other patient characteristics are critically important: heart failure, age, and frailty [8]. Importantly, risk increased with cumulative comorbidity burden; in a population-based cohort of 31,056 ACS patients, the presence of three or more of the examined comorbidities was associated with an almost threefold higher risk of MACE compared with no comorbidities (aHR 2.61; 95% CI 2.40–2.85) [7]. The following table summarizes the quantitative risk associated with these key independent predictors, based primarily on Zhai et al. [28], except for primary heart failure, which is derived from Okkonen et al. [18].

From a clinical point of view, it's important to underline the severity of the ACS event itself. Some data suggest that NSTEMI patients may have higher long-term mortality and MACE, which is often attributed to their typically older age and greater burden of comorbidities [7]. Conversely, other large studies identify STEMI presentation as a major predictor of MACE [8]. The BleeMACS registry provided a more detailed view: both STEMI and NSTEMI patients with multivessel disease had very high MACE rates if incompletely revascularized (24.6% and 13.9%, respectively), indicating that the extent of underlying disease and the adequacy of treatment are critical drivers of risk, regardless of the initial ECG presentation [20]. There are also very important prognostic markers influencing the rate of MACE after ACS: in a large contemporary post-PCI cohort, cardiogenic shock and cardiac arrest were among the strongest predictors of subsequent MACE, with adjusted hazard ratios of 2.12 (95% CI 2.08–2.16) and 1.67 (95% CI 1.63–1.70), respectively [8]. In a separate analysis, Killip class greater than II was associated with an odds ratio of 2.39 for post-PCI MACE [28]. Left Ventricular Ejection Fraction: LVEF of 40% or less is a strong predictor of subsequent MACE, with an OR of 1.86 [28]. Angiographic and procedural findings: multivessel coronary artery disease is a consistent marker of a high atherosclerotic burden and is strongly associated with worse outcomes. TIMI (Thrombolysis in Myocardial Infarction) Flow Grade: A final TIMI flow grade of II or less indicates suboptimal reperfusion at the epicardial level and is associated with a higher risk of MACE (OR 1.41) [28]. Importantly, substantial residual modifiable risk may persist after ACS; in the Finnish cohort, 23% of survivors continued smoking, and 24% had cholesterol levels >5 mmol/L, despite 86% receiving lipid-lowering medication [18].

Adherence to Guideline-directed medical and interventional therapies is the cornerstone of improving outcomes after ACS:

1. Revascularization strategy: A growing body of evidence from randomized trials and meta-analyses shows that a strategy of complete revascularization (treating both the culprit lesion and significant non-culprit lesions) is superior to a "culprit-only" PCI strategy. This approach is associated with significantly lower rates of long-term MACE in both STEMI and NSTEMI patients [14,20]. Furthermore, registry data show that receiving revascularization within 28 days of the event is a protective factor, associated with an OR of 0.82 for MACE at 3 years [18].

2. The use of high-intensity statin therapy (e.g., atorvastatin 80 mg, rosuvastatin 20-40 mg) is a Class 1 recommendation in all major guidelines for ACS patients without contraindications [6,14]. A comprehensive meta-analysis involving over 26,000 patients demonstrated that high-intensity statin therapy provides a significant reduction in MACE compared to standard- or moderate-intensity regimens, with a relative risk (RR) of 0.77 [27]. Another meta-analysis found that intensive statin therapy reduced MACE (OR 0.84) and also reduced all-cause mortality in the post-ACS setting.[1] The benefit appears to be time-dependent, with the most significant risk reduction emerging after 12 months of continuous treatment [27].

3. Dual Antiplatelet Therapy (DAPT) Duration: The optimal duration of DAPT (aspirin plus a P2Y12 inhibitor) is one of the most important topics in post-ACS care, representing a risk/benefit balance between reducing ischemic risk and increasing bleeding risk. A meta-analysis of trials examining DAPT beyond one year found that extended therapy reduced ischemic events, including cardiovascular death, MI, and stroke, but at the cost of a significant increase in major bleeding [25]. Meta-analyses have compared shorter DAPT durations (e.g., 1-3 months or 3-6 months) to the standard 12-month regimen, particularly in patients treated with newer-generation drug-eluting stents. These analyses consistently show that shorter DAPT is associated with a significantly lower risk of major bleeding without a statistically significant increase in the composite endpoint of MACE or its individual ischemic components like MI or stent thrombosis [12]. The optimal duration for an individual patient should be determined by a careful assessment of their competing risks for ischemic events (e.g., high-risk features like diabetes, multivessel disease, prior MI) versus their risk for bleeding (e.g., advanced age, renal failure, history of bleeding). This individualized (personalized) approach is the new paradigm for secondary prevention.

4. SGLT2 inhibitors: Given the prognostic importance of heart failure after ACS, the role of SGLT2 inhibitors early after myocardial infarction has been evaluated in the DAPA-MI and EMPACT-MI trials. DAPA-MI demonstrated improvement in a hierarchical cardiometabolic outcome with dapagliflozin, although no clear reduction in cardiovascular death or heart-failure hospitalization was observed [10]. Similarly, EMPACT-MI did not significantly reduce the primary composite of heart-failure hospitalization or all-cause death, although first heart-failure hospitalizations were reduced with empagliflozin [3]. A subsequent meta-analysis of 16 randomized and observational studies encompassing over 130,000 patients found that initiation of SGLT2 inhibitors after ACS was associated with a significant reduction in all-cause mortality (HR 0.77; 95% CI 0.67–0.89) and cardiovascular mortality (HR 0.83; 95% CI 0.70–0.99); subgroup analysis showed a significant reduction in all-cause mortality (HR 0.73; 95% CI 0.62–0.86) and recurrent

Table 1. *Timing of MACE after ACS.*

Follow-up Period	Reported MACE Rate (%) (95% CI)	Source (Study Name/Type)	Study Type	Notes
30 Days	5.0% - 11.2%	(Turkish AMI cohort) [5]	Observational Cohort MACE composition: mortality, nonfatal myocardial infarction, nonfatal stroke, acute heart failure, ischemia-driven revascularization, major bleeding, and acute renal failure developed within 30 days.	Rate varied by timing of revascularization strategy.
90 Days	4.1% (3.0 - 5.7)	(Chi et al) [4]	Meta-analysis of 7 RCTs	Pooled rate from high-risk clinical trial populations. Represents ~49% of 1-year events.
6 Months	5.8% - 6.6%	(CPACS-3 Trial) [26]	Large Randomized Trial (China) MACE composition: all-cause mortality, nonfatal myocardial infarction, and nonfatal stroke.	Provides a specific benchmark from a large trial in a non-Western population.
12 Months	3.6%	(Hue Central Hospital) [15]	Single-Center Cohort (Vietnam)	Low rate observed in a small, single -center post-PCI cohort (n=337).
12 Months	8.3% (7.1 - 9.8)	(Chi et al.) [4]	Meta-analysis of 7 RCTs	Pooled rate from high-risk clinical trial populations.
12 Months	34.4%	(Finnish Registry) [18]	National Registry	Broad MACE definition included recurrent ACS, heart failure, stroke, cardiovascular events, and all-cause death.
12 Months	20.9% to 53.1%	(UK Frailty Study) [21]	National Registry (UK)	Demonstrates the profound impact of frailty on MACE, with rates more than doubling from low- to high-frailty groups.

Table 2. *Main risk factors of MACE.*

Risk Factor	Associated Risk Metric	Value	95% Confidence Interval (CI)	Significance / Note
Killip Class > II	Odds Ratio (OR)	2.39	2.17 - 2.64	Strongest patient-related predictor in the meta-analysis, indicating severe hemodynamic compromise.
History of Smoking	Odds Ratio (OR)	2.08	1.51 - 2.85	A powerful and modifiable risk factor.
Primary Heart Failure	Odds Ratio (OR) for 3-year MACE	1.98	1.67 - 2.36	Indicates significant myocardial damage from the index event.
LVEF ≤40%	Odds Ratio (OR)	1.86	1.71 - 2.03	Key indicator of poor left ventricular function and prognosis.
Renal Insufficiency	Odds Ratio (OR)	1.82	1.37 - 2.43	Highlights the cardio-renal connection and systemic nature of risk.
Diabetes Mellitus	Odds Ratio (OR)	1.70	1.43 - 2.02	A major metabolic driver of atherosclerosis and adverse outcomes.
Hypertension	Odds Ratio (OR)	1.62	1.35 - 1.96	A common and significant contributor to long-term risk.
Dyslipidemia	Odds Ratio (OR)	1.50	1.19 - 1.89	The fundamental substrate for the atherosclerotic process.

myocardial infarction (HR 0.83; 95% CI 0.69–0.99) among patients with type 2 diabetes, while no significant effect was observed in patients without diabetes [23]. Thus, while SGLT2 inhibitors may provide benefit in selected post-MI patients, evidence for a specific reduction in post-ACS MACE remains limited.

5. Given the contribution of diabetes and chronic kidney disease to post-ACS cardiovascular risk, GLP-1 receptor agonists have demonstrated cardiovascular benefit in high-risk metabolic populations. In the SOUL trial, oral semaglutide significantly reduced 3-point MACE in patients with type 2 diabetes and established atherosclerotic cardiovascular disease, chronic kidney disease, or both (HR 0.86; 95% CI 0.77–0.96) [17]. However, direct post-ACS evidence is less convincing: in ELIXA, lixisenatide did not significantly reduce cardiovascular events in patients with type 2 diabetes and a recent ACS (HR 1.02; 95% CI 0.89–1.17) [19]. A scoping review of ten studies evaluating exenatide and liraglutide in adults with STEMI found GLP-1 receptor agonist use to be safe and well tolerated; exenatide was associated with improvements in infarct size, myocardial salvage, and cardiac function (though two trials in broader STEMI populations reported no post-infarction

improvement), and liraglutide was associated with improved myocardial salvage, infarct size, left ventricular ejection fraction, stroke volume, and no-reflow across three trials, supported by favorable biomarker changes, though without significant reductions in major cardiovascular events [22]. These findings support GLP-1 receptor agonists primarily as part of long-term secondary prevention in appropriately selected high-risk patients.

6. Colchicine: The early inflammatory response described above as a driver of the 90-day vulnerable period has itself become a treatment target: low-dose colchicine has been investigated as a strategy to reduce recurrent cardiovascular events after myocardial infarction by targeting this residual inflammation. In COLCOT, low-dose colchicine significantly reduced the composite cardiovascular endpoint compared with placebo (HR 0.77; 95% CI 0.61–0.96) [24]. However, the more recent CLEAR SYNERGY trial found no reduction in cardiovascular death, recurrent MI, stroke, or ischemia-driven revascularization (HR 0.99; 95% CI 0.85–1.16) [11]. These contrasting findings indicate that the role of colchicine in routine post-ACS secondary prevention remains uncertain.

The comprehensive analysis of MACE incidence, timing, and predictors following an acute coronary syndrome provides a clear mandate for refining clinical practice and focusing future research efforts. The evidence points toward the need for more intensive, personalized, and front-loaded secondary prevention strategies to mitigate the substantial residual risk that persists despite modern therapies.

Discussion.

Despite significant advances, critical evidence gaps remain. Addressing these gaps is essential for further improving patient outcomes:

1. The single greatest issue in synthesizing evidence in this field is the lack of a standardized, universally accepted definition for MACE [2]. International cardiovascular societies, such as the ESC and AHA/ACC, should lead a consensus initiative to develop and promote standardized composite endpoint definitions for use in both clinical trials and observational research. This would substantially improve the comparability, transparency, and clinical utility of future studies.

2. Future research should include trials specifically designed to test novel interventions targeting the unique pathophysiology of the first 90 days post-ACS. Therapies targeting acute inflammation, residual thrombotic risk, or early adverse ventricular remodeling should be evaluated using a primary endpoint of 90-day MACE, as exemplified by the design of the AEGIS-II trial [4].

3. While current risk scores are valuable, there is a clear need for more sophisticated and dynamic prediction models. In one single-center comparison, RISK-PCI demonstrated notably stronger discrimination for 30-day MACE (AUC 0.94) than admission or discharge GRACE scores (AUC 0.73 and 0.65, respectively) [9]. However, given the retrospective single-center design and relatively small sample size, these findings require confirmation in larger external cohorts before broader clinical application. Future models should leverage large, diverse, real-world datasets to provide more accurate and personalized risk stratification [16]. These models should be dynamic, allowing for the integration of new data points (e.g., biomarker results, medication adherence) over time to update a patient's risk profile continuously.

Limitations.

As a narrative synthesis, this review integrates evidence from studies with diverse designs, populations, follow-up periods, and MACE definitions. This heterogeneity limits direct comparison of event rates and treatment effects across studies and should be considered when interpreting the available evidence. Although priority was given to contemporary, high-quality publications and major guidelines, the narrative approach does not provide the exhaustive study identification characteristic of a systematic review.

Conclusion.

While the management of ACS has improved dramatically, the post-discharge period remains a time of significant hazard. By recognizing the temporal pattern of risk, personalizing therapy based on a comprehensive assessment of individual patient characteristics, and focusing research on the most pressing evidence gaps, the clinical and scientific communities can continue to improve the long-term prognosis for survivors of acute coronary syndromes.

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GEORGIAN.

kvlevis mizani: winamdebare mimoxilva ajamebs MACE-is ganmartebebis, maTi ACS-is Semdgomi ganviTarebis ZiriTadi ganmapirobebeli risk-faqtorebis da mtkicebulebebze dafuZnebuli riskis Semcirebis strategiebis Sesaxeb arsebul mtkicebulebebs, aseve gamoavlens ZiriTad meTodologiur xarvezebs.

masala da meTodebi: PubMed-isa da jvaredini wyaroebis mqone saxelmZRvanelo dokumentebis meSveobiT identificirebuli randomizebuli kontrolirebadi kvlevebis, metaanalizebis, didi reestrebisa da dakvirvebiTi kvlevebis narativiuli mimoxilva, romlebSic Seswavlilia mwvave koronaruli sindromis Semdgomi MACE-i. Ziebis sistemuri protokoli an raodenobrivi analizi ar ganxorcielebula; upiratesoba mieniWa recenzirebul publikaciebs, moqmed gaidlainebsa da maRali impaqt-faqtoris mqone meta-analizebs, xolo Semajamebeli SefasebebisTvis ki ganxilulia originaluri wyaroebi.

Sedegebi: MACE-is ganmartebebi heterogenulia. klasikuri 3-komponentiani kombinacia (kardiovaskuluri sikvdili, arafataluri miokardiumis infarqti, arafataluri insulti) warmoadgens miRebul standarts, Tumca xSirad gvxvdeba 4- da 5-komponentiani versiebi, romlebic moicavs revaskularizacias, arastabilur stenokardias an gulis ukmarisobis gamo hospitalizacias, xolo administraciuli monacemTa bazebSi kardiovaskulur sikvdilianobas xSirad anacvleben yvela mizeziT gamowveuli sikvdilianobiT. ufro farTo ganmartebebi zrdis arasasurvel movlenaTa maCveneblebs da SeiZleba gavlena moaxdinos mkurnalobis Sedegebis interpretaciazec. erT-erTi didi metaanalizis monacemebi miuTiTebs, rom pirveli wlis ganmavlobaSi arasasurveli movlenebis TiTqmis naxevari 90 dRis ganmavlobaSi viTardeba, 90-dRiani M MACE maCvenebeli ki saerTo jamSi 4.1% Seadgens. Tumca, unda aRiniSnos, rom erTwliani garTulebebis sixSire meryeobs 3.6%-dan dabali riskis mqone pacientebis jgufSi 34.4%-de did erovnul reestrSi, rac asaxavs Seswavlil pacientTa klinikuri maxasiaTeblebis, Catarebuli revaskularizaciis sisrulisa da MACE Semadgeneli komponentebis gansxvavebebs. ACS-is Semdgomi MACE-is ganviTarebis yvelaze Zlieri prediqtorebi aRmoCnda daavadebis klinikuri gamovlinebis simZime (Killip-is klasi >II, kardiogenuli Soki/gulis gaCereba, marcxena parkuWis gandevnis fraqcia (LVEF) ≤40%), Tanmxlebi daavadebebis arseboba (Tirkmlis qronikuli daavadeba,

Saqriani diabeti, hipertenzia, dislipidemia, Tambaqos moweva, asaki/mowyvladoba) da anatomiuri/proceduruli faqtorebi (mravalsisxlZarRvovani dazianeba, arasruli revaskularizacia, intervenciis Semdgomi suboptimaluri TIMI nakadi). sruli revaskularizacia, maRali intensivobis statinebiT Terapia, ormagi antiagregantuli Terapiis individualizebuli xangrZlivoba da adreuli intensiuri meoradi prevencia TiToeul SemTxvevaSi dakavSirebuli iyo MACE-is SemcirebasTan.

daskvna: mwvave koronaruli sindromis (ACS) Semdgomi arasasurveli gul-sisxlZarRvTa movlenebis (MACE) umetesoba gaweridan 90 dRis ganmavlobaSi vlindeba. Tumca, maTi sixSire mniSvnelovnad gansxvavdeba saboloo wertilebis ganmartebebis heterogenulobisa da Seswavlil populaciebSi arsebuli gansxvavebebis gamo. MACE ganmartebebisa da Sefasebis kriteriumebis standartizacia da adreul saboloo wertilebze orientirebuli kvlevebis Catareba, mniSvnelovnad gaaumjobesebs axali mtkicebulebebis sinTezs. klinicistebma prioriteti unda mianiWon srul revaskularizacias, optimizebul lipidebis damwev Terapias, individualur antiTrombozul Terapias da adreul, intensiur meorad prevencias.

sakvanZo sityvebi: mwvave koronaruli sindromi, arasasurveli kardiovaskuluri movlenebi, MACE, kombinirebuli saboloo wertilebi, vadebi, risk-faqtorebi, meoradi prevencia, naratiuli mimoxilva.

РУССКИЙ.

Цель исследования: В представленном обзоре обобщаются имеющиеся данные об определениях MACE, сроках их возникновения и величине риска развития после ACS , основных провоцирующих факторах и научно обоснованных стратегиях снижения риска развития, а также выявляются ключевые методологические пробелы.

Материалы и методы: Нарративный обзор рандомизированных контролируемых исследований, метаанализов, крупных регистров и наблюдательных исследований, сообщающих о серьезных сердечно-сосудистых событиях после ACS, выявленных с помощью PubMed и перекрестных ссылок на современный гайдлайны. Систематический протокол поиска или количественный анализ не производились; приоритет отдавался рецензируемым публикациям, действующим руководствам и высокоэффективным метаанализам, при этом для обощающих оценок цитировались оригинальные источники.

Результаты: Определения серьезных сердечно-сосудистых событий (MACE) неоднородны. Классическая трехкомпонентная комбинация (сердечно-сосудистая смерть, нефатальный инфаркт миокарда, нефатальный инсульт) является основным стандартом, но также распространены 4- и 5-компонентные версии, включающие реваскуляризацию, нестабильную стенокардию или госпитализацию по поводу сердечной недостаточности, а в административных базах данных часто вместо сердечно-сосудистой смертности используется общая смертность. Более широкие определения завышают частоту событий и могут влиять на интерпретацию эффектов лечения. Данные одного из крупнейших метаанализов указывают , что почти половина нежелательных событий в течение первого года происходит в течение 90 дней, при этом общая частота MACE за 90 дней составила 4,1%. Хотя необходимо отметить, что показатели за один год варьируют от 3,6% в небольшой когорте пациентов с более низким риском до 34,4% в крупном национальном регистре, что отражает различия в составе пациентов, полноценности проведенной реваскуляризации и определениях компонентов MACE . Наиболее сильными предикторами серьезных сердечно-сосудистых событий после острого коронарного синдрома были клиническая тяжесть индекс ACS (класс Киллипа >II, кардиогенный шок/остановка сердца, фракция выброса левого желудочка ≤40%), сопутствующие заболевания (хроническая болезнь почек, диабет, гипертония, дислипидемия, курение, возраст/слабость) и анатомические/процедурные факторы (многососудистое поражение, неполная реваскуляризация, достигнутый при реваскуляризации субоптимальный кровоток TIMI). Полная реваскуляризация, терапия статинами высокой интенсивности, индивидуализированная продолжительность двойной антиагрегантной терапии и структурированная ранняя вторичная профилактика были связаны со снижением частоты серьезных сердечно-сосудистых событий.

Выводы: Большинство серьезных сердечно-сосудистых событий (MACE) после острого коронарного синдрома (ACS) наблюдается в течение 90 дней после выписки. Однако их частота значительно варьирует из-за неоднородности определений конечных точек и различий в изученных популяциях. Стандартизация определения MACE и критериев их оценки, а также проведение исследований, рассчитанных на ранние конечные точки, существенно улучшат синтез доказательств. Клиницистам следует отдавать приоритет полной реваскуляризации, оптимизированной липидоснижающей терапии, индивидуализированной антитромботической терапии и ранней интенсивной вторичной профилактике.

Ключевые слова: Острый коронарный синдром, серъезные нежелательные сердечно-сосудистые события (MACE), комбинированные конечные точки, сроки, риск-факторы, вторичная превенция, нарративный обзор.