

ACUTE CORONARY SYNDROME: EARLY DIAGNOSIS AND PROGNOSTIC IMPACT

GABRIEL FAGUNDES MOREIRA¹, PAULO VINÍCIUS DIAS¹, MATHEUS LÚCIO FERNANDES GOMES¹, IGOR DINIZ ALVES¹,
AIRTON SILVA DE SANTANA¹

1. Graduando em Medicina na Universidade Federal de Goiás, Goiânia/GO, Brazil.

ABSTRACT

Acute coronary syndrome remains one of the leading causes of cardiovascular morbidity and mortality and requires early diagnosis, accurate risk stratification, and rapid therapeutic decision-making in emergency settings. This study aimed to analyze the available evidence on early diagnostic strategies and their prognostic implications in patients with suspected or confirmed acute coronary syndrome. This is an integrative literature review conducted in the PubMed/MEDLINE database, including five studies published between 2016 and 2026. The findings show that high-sensitivity cardiac troponin has expanded the ability to identify myocardial infarction and different categories of myocardial injury, although this diagnostic improvement has not consistently translated into better clinical outcomes. The HEART score showed superior performance compared with TIMI and GRACE in identifying low-risk patients, supporting safer hospital discharge strategies. Coronary computed tomography angiography demonstrated operational value by reducing unnecessary invasive procedures but without a proven impact on mortality or nonfatal myocardial infarction. It is concluded that prognosis in acute coronary syndrome depends not only on diagnostic accuracy, but also on the integration of biomarkers, clinical risk scores, imaging methods, and therapeutic protocols directed at the newly identified high-risk patient groups.

Keywords: Acute coronary syndrome, Biomarkers, Cardiac troponin, Early diagnosis, Prognosis.

INTRODUCTION

Acute coronary syndrome (ACS) represents one of the leading causes of morbidity and mortality worldwide and constitutes a daily challenge in emergency departments. The assessment of patients presenting with acute chest pain requires speed and accuracy, since early diagnosis and the immediate initiation of evidence-based therapies are decisive for preserving cardiac muscle and patient survival. Historically, the focus of initial care has been the rapid exclusion of acute myocardial infarction (AMI) and the identification of patients requiring immediate invasive intervention, balancing the safety of hospital discharge with the rational use of healthcare system resources.

In recent decades, the diagnostic paradigm of ACS has undergone a profound transformation with the introduction of high-sensitivity cardiac troponins (hs-cTn). The ability of these biomarkers to detect minute

concentrations of myocardial protein has not only accelerated algorithms for ruling out (rule-out) and confirming (rule-in) ischemia, but has also driven the reclassification of myocardial injury in accordance with the Fourth Universal Definition of Myocardial Infarction. In parallel with laboratory advances, the systematic application of validated clinical risk scores, such as History, ECG, Age, Risk factors, and Troponin (HEART), Thrombolysis in Myocardial Infarction (TIMI), and the Global Registry of Acute Coronary Events (GRACE), has become a cornerstone in structuring clinical reasoning. The combination of these tools aims to reduce medical subjectivity, optimizing predictive accuracy for short-term major adverse cardiac events (MACE).^{1,2}

In addition to laboratory and clinical approaches, the initial diagnostic armamentarium has been expanded by incorporating noninvasive imaging tests. Coronary computed tomography angiography (CTCA) has emerged as a complementary strategy for intermediate-risk patients, intended to function as an anatomical filter capable of confirming or ruling out the presence of obstructive coronary artery disease, with the aim of reducing the incidence of unnecessary diagnostic catheterizations.³ Additionally, recent evidence has broadened the prognostic spectrum of initial assessment, demonstrating that even detectable baseline concentrations of hs-cTn, although still within reference limits, are directly correlated with the future development of ventricular failure, suggesting the feasibility of risk prediction extending beyond the acute phase of the ischemic event.⁴

However, contemporary literature reveals a significant paradox between advances in diagnostic accuracy and actual modification of clinical prognosis (Knowledge Gap). Although the integration of hs-cTn with emergency-oriented clinical scores, such as HEART, has improved the safety of discharging very-low-risk patients, increased laboratory sensitivity has generated an epidemic of diagnoses of type 2 myocardial infarction and acute and chronic non-atherosclerotic myocardial injury.⁵ These subgroups, which currently account for a substantial proportion of troponin elevations, have cardiovascular and noncardiovascular mortality rates equivalent to or higher than those of type 1 myocardial infarction. However, the increased recognition of these etiologies has not been accompanied by a corresponding adaptation in secondary prevention and treatment protocols.⁵ These patients systematically receive fewer cardioprotective therapies and appropriate interventions, resulting in stagnant outcomes.

Likewise, the benefit of early anatomical screening with CTCA appears to be restricted to purely operational aspects of hospital workflow, lacking substantial evidence regarding reduction in hard outcomes (mortality or nonfatal myocardial infarction) among intermediate-risk populations.³

Therefore, there is a critical need to understand the limitations of the current application of stratification tools. Technological advances have provided an unprecedented level of diagnostic and anatomical detail; however, the absence of structured therapeutic pathways for the new risk categories identified by hs-cTn and the uncertainties regarding the isolated impact of imaging tests highlight fragmentation in patient care. It is imperative to critically analyze how highly precise diagnosis is, or is not, being translated into risk modification, in order to guide future therapeutic strategies aimed at reducing morbidity and mortality in this complex population.

OBJECTIVES

Analyze the evidence available in the literature on early diagnostic strategies and their prognostic consequences in patients with suspected or confirmed Acute Coronary Syndrome.

METHODS

This is an integrative literature review. The search was conducted in the PubMed/MEDLINE database using controlled MeSH descriptors and free-text terms: ("Acute Coronary Syndrome" OR "STEMI" OR "NSTEMI" OR "unstable angina") AND ("early diagnosis" OR "troponin" OR "high-sensitivity troponin" OR "HEART score" OR "GRACE score" OR "cardiac biomarkers") AND ("prognosis" OR "mortality" OR "outcomes" OR "MACE"). Filters were applied for studies published between 2016 and 2026. The search strategy yielded 68 articles, from which 5 studies with the greatest methodological relevance and suitability to the topic were selected.

RESULTS AND DISCUSSIONS

This integrative review selected five studies published between 2017 and 2023, with different methodological designs—including randomized clinical trials, multicenter prospective cohorts, and a systematic review—totaling more than 77,000 patients evaluated in emergency or inpatient settings for suspected acute coronary syndrome (ACS). The studies were conducted in the United Kingdom^{3,4,5} the Netherlands^{1,2} Canada¹, the United States¹, Israel¹, Switzerland¹, and Australia¹, covering different early diagnostic strategies: high-sensitivity cardiac troponin (hs-cTn), clinical risk scores, coronary computed tomography angiography (CTCA), and diagnostic classification based on the Universal Definition of Myocardial Infarction. The characteristics of the included studies are summarized in Table 1.

Table 1: Characteristics of the Included Studies.

Study	Design	N	Country	Primary outcome	Follow-up
Poldervaart et al., 2017 ²	Prospective multicenter cohort	1,748	Netherlands	MACE at 6 weeks	6 weeks
O’Rielly et al., 2023 ¹	Systematic review	20,959	Multicenter	MACE at 30 days	30 days
Chapman et al., 2020 ⁵	Stepped-wedge RCT (High-STEACS)	48,282	United Kingdom	IAM or CV death at 1 year	1 year
Stelzle et al., 2018 ⁴	Prospective cohort	4,748	United Kingdom	Hospitalization for IC	~5 months
Gray et al., 2021 ³ -CTCA	Multicenter RCT (RAPID)	1,748	United Kingdom	Death or MI at 1 year	1 year

Source: Prepared by the authors, 2026.

Clinical Risk Stratification Using Validated Scores

The direct comparison of clinical risk scores in patients presenting with chest pain in the emergency department was addressed in a complementary manner by Poldervaart et al.² and O’Rielly et al.¹, providing perspectives that converge on the superiority of the HEART score over the other available tools.

Poldervaart et al.² conducted a multicenter prospective cohort study in 9 hospitals in the Netherlands, including 1,748 patients evaluated for chest pain, excluding cases of immediately recognized STEMI.

The primary outcome—MACE at 6 weeks, including unstable angina, NSTEMI, STEMI, coronary stenosis managed conservatively, revascularization, and cardiovascular death—occurred in approximately 19% of the sample (325–326 patients). The HEART score demonstrated superior discriminatory performance compared with TIMI and GRACE, with areas under the ROC curve (AUC) of 0.86 (95% CI 0.84–0.88), 0.80 (95% CI 0.78–0.83), and 0.73 (95% CI 0.70–0.76), respectively, with all paired differences being statistically significant ($p < 0.001$)². Using a minimum sensitivity criterion of 95%, the HEART score with a cutoff ≤ 3 classified 40.5% of patients as low risk, with a residual MACE rate of 2.0% and a negative predictive value (NPV) of 98% (95% CI 97–99%).² The TIMI score identified 25.1% of patients as low risk, and GRACE only 19.1%, with residual MACE rates of 3.2% and 3.6%, respectively.² In the more conservative scenario, with a minimum sensitivity of 98%, the HEART score with a cutoff ≤ 2 classified 21.8% of patients as low risk, with a residual MACE rate of only 0.8%, no deaths, and an NPV of 99% (95% CI 98–100%).² The TIMI score was unable to identify a low-risk group at this level of safety, and GRACE classified only 13.2% of patients, with a residual MACE rate of 2.2%.²

These findings are supported and complemented by the systematic review by O’Rielly et al.¹, which evaluated the prognostic performance of clinical scores specifically in patients in whom myocardial infarction had already been ruled out by hs-cTn, totaling 20,959 patients across six eligible studies. In this post-MI rule-out population, the HEART score with a cutoff ≤ 3 , combined with hs-cTnT, was the score that classified the largest proportion of patients as low risk—up to 62.5% in one study—with sensitivity for 30-day MACE ranging from 93.8% to 100% and NPV between 99.7% and 100%.¹ The TIMI score ≤ 1 classified similar proportions of patients as low risk in some studies, but with highly variable sensitivity across cohorts (66.7% to 98.9%), limiting its reliability as a universal triage tool.¹ The GRACE score with a cutoff < 73 demonstrated unsatisfactory performance in this population, achieving a sensitivity of only 75.4%¹—a finding that reflects the inadequacy of a tool originally developed for populations with established ACS when applied to patients who had already undergone hs-cTn algorithms. The performance hierarchy identified—HEART superior to TIMI and GRACE—was consistent across both studies, regardless of the populations and comparative methods used.^{1,2}

A finding of particular clinical relevance concerns the limited incremental value of the scores after exclusion of AMI by hs-cTn¹. The 30-day MACE rate in this population is already intrinsically low—below 1% in most studies—reducing the margin for additional gain from any extra score. This observation suggests that patients with negative hs-cTn results according to a validated algorithm may be candidates for safe discharge without the need for routine noninvasive testing, regardless of individual clinical characteristics¹.

Impact of High-Sensitivity Troponin on Diagnosis and Prognosis

Chapman et al.⁵ demonstrated, in a multicenter stepped-wedge randomized clinical trial involving 48,282 consecutive patients with suspected ACS across 10 hospitals in Scotland, that implementation of hs-cTnI significantly increased recognition of different categories of myocardial injury compared with conventional troponin. The increases were 11% for the diagnosis of type 1 myocardial infarction (510/4,471), 22% for type 2 MI (205/916), 36% for acute myocardial injury (443/1,233), and 43% for chronic myocardial injury (389/898)⁵. This pattern shows that the diagnostic gain provided by hs-cTn is disproportionately greater for non-atherosclerotic categories of myocardial injury, with direct implications for prognostic stratification in a subgroup of patients that is usually underdiagnosed.

Diagnostic classification according to the Fourth Universal Definition of Myocardial Infarction revealed that all identified categories of myocardial injury confer significantly increased cardiovascular

risk at 1 year, regardless of pathogenesis.⁵ The risk of myocardial infarction or cardiovascular death was highest in patients with type 1 AMI (csHR 5.64; 95% CI 5.12–6.22), but remained substantial in the other categories: type 2 AMI (csHR 3.50; 95% CI 2.94–4.15), acute myocardial injury (csHR 4.38; 95% CI 3.80–5.05), and chronic myocardial injury (csHR 3.88; 95% CI 3.31–4.55), all compared with patients without myocardial injury.⁵ The risk of noncardiovascular death was highest in patients with acute myocardial injury (csHR 2.65; 95% CI 2.33–3.01), a category in which more than one third of deaths occurred within the first 30 days due to pneumonia, exacerbation of chronic obstructive pulmonary disease, or sepsis—extracardiac causes whose management requires a multidisciplinary approach.

Despite the demonstrated diagnostic and prognostic capacity, implementation of hs-cTn did not translate into consistent improvement in clinical outcomes⁵. There was no reduction in the primary outcome in any of the diagnostic categories after the introduction of hs-cTnI, including type 1 AMI, in which modest increases in antiplatelet therapy (43% vs. 61%) and coronary revascularization (35% vs. 44%) were observed, without an impact on the composite event (csHR 1.00; 95% CI 0.82–1.21).⁵ For patients with type 2 AMI and myocardial injury, there was no change in investigation, treatment, or outcomes after implementation⁵, exposing a critical gap: the diagnostic advance provided by hs-cTn was not accompanied by appropriate therapeutic protocols for these populations. Patients with type 2 AMI underwent coronary angiography in only 10% of cases and percutaneous intervention in only 2%, compared with 59% and 41%, respectively, among patients with type 1 MI⁵, despite presenting a 1-year cardiovascular risk more than 3 times higher than that of patients without myocardial injury.

Prognostic Value of hs-cTnI Beyond the Diagnosis of Myocardial Infarction

Stelzle et al.⁴ broadened the prognostic scope of hs-cTnI by demonstrating, in a prospective cohort of 4,748 consecutive patients with suspected ACS, that hs-cTnI concentration independently predicts the risk of hospitalization for heart failure—even among patients with troponin levels within the normal reference range. The relationship between hs-cTnI concentration and the risk of hospitalization for heart failure was nonlinear: each doubling of hs-cTnI concentration below the upper reference limit (URL) was associated with a 2.80-fold increase in the risk of hospitalization for heart failure (HR 2.80; 95% CI 1.81–4.31), whereas above the URL, higher concentrations did not confer a significant additional risk (HR 1.03; 95% CI 0.96–1.12).⁴ Incidence rates of hospitalization for heart failure ranged from zero in the two lowest troponin quartiles to 118 per 1,000 person-years in the group with troponin levels above the URL, with progressively higher adjusted HRs of 3.0 (Group 3), 11.7 (Group 4), and 28.9 (Group 5) compared with the reference group⁴.

The discriminatory ability of hs-cTnI for predicting heart failure was comparable to that of a complete clinical model (C-statistic 0.80 for both), and its combination with clinical variables increased discrimination to 0.86 (95% CI 0.82–0.89; $p < 0.001$)⁴, a level indicating usefulness for individual event prediction. Patients in the two lowest troponin quartiles experienced no hospitalization for heart failure or cardiac death during follow-up, reinforcing the negative predictive value of hs-cTn and the safety of early discharge strategies for this subgroup⁴. These data are consistent with the findings of O’Rielly et al.¹ and Poldervaart et al.² regarding the excellent performance of negative hs-cTn results in identifying very-low-risk patients.

Role of Early Coronary Computed Tomography Angiography

Gray et al.³ evaluated, in the RAPID-CTCA trial, whether the early incorporation of CTCA into standard care could improve clinical outcomes in 1,748 patients with suspected ACS and at least one intermediate-

risk marker—elevated troponin (57%), abnormal ECG (61%), or previous coronary artery disease (34%)—with a mean GRACE score of 115. The primary outcome of all-cause death or nonfatal type 1 or 4b myocardial infarction at 1 year occurred in 5.8% of patients in the CTCA group and 6.1% in the control group (adjusted HR 0.91; 95% CI 0.62–1.35; $p=0.65$)³, indicating no significant clinical benefit of the strategy on hard outcomes. All secondary outcomes related to mortality and cardiovascular events were likewise neutral, with no significant heterogeneity in subgroup analyses by age, sex, GRACE score, history of coronary artery disease, or troponin³.

The benefit of CTCA was concentrated on the care process: there was a significant reduction in the rate of invasive coronary angiography (54.0% vs. 60.8%; adjusted HR 0.81; 95% CI 0.72–0.92; $p=0.001$) and in subsequent noninvasive testing for coronary artery disease or ischemia (19.4% vs. 26.2%; adjusted HR 0.66; 95% CI 0.54–0.81; $p<0.001$)³. There was no difference in revascularization (34.2% vs. 33.1%; $p=0.76$) or in pharmacological treatment for ACS (67.8% vs. 66.6%; $p=0.63$)³. The anatomical findings of CTCA provide a relevant interpretive perspective: 23% of patients had normal coronary arteries, 29% had nonobstructive disease, and 47% had obstructive disease³—showing that more than half of intermediate-risk patients, even with abnormal biomarkers or ECG findings, did not have significant coronary obstruction, which supports the role of CTCA as an anatomical triage tool to avoid unnecessary invasive angiography in selected subgroups. CTCA was, however, associated with a slight increase in length of stay (2.2 vs. 2.0 days; $p=0.009$)³, differing from studies in low-risk populations, in which the examination usually reduces hospital stay.

Integration of Findings and Clinical Implications

The integrative analysis of the five studies allows a coherent overview of early diagnosis and prognostic impact in ACS to be outlined. hs-cTn constitutes the central pillar of the contemporary diagnostic strategy^{4,5} with greater ability than previous generations of troponin to identify myocardial injury in its different forms.⁵ However, the diagnostic gain did not automatically translate into improved outcomes⁵, demonstrating that the clinical value of hs-cTn lies primarily in risk stratification—both to identify patients who require immediate intervention and to safely characterize those at very low risk who are eligible for early discharge.^{1,4}

In this context, the application of validated clinical scores represents an essential complementary step. The HEART score consistently demonstrated superiority over TIMI and GRACE both in event discrimination (AUC 0.86 vs. 0.80 vs. 0.73)² and in the efficient identification of low-risk patients after MI was ruled out by hs-cTn (up to 62.5% classified as low risk, with an NPV of 99.7% to 100%).¹ This superiority may be attributed to the alignment of the HEART score with the specific clinical setting of the emergency department—having been developed for patients with undifferentiated chest pain—unlike GRACE and TIMI, which are more strongly oriented toward prognostic stratification in patients with already confirmed ACS.² The integration of hs-cTn and structured clinical scores such as HEART therefore represents the most rational approach to risk stratification in ACS, combining the analytical precision of the biomarker with the multivariable clinical context of the score.^{1,2}

The prognostic impact of the different categories of myocardial injury identified by hs-cTn represents the most relevant clinical gap highlighted by this review. Type 2 AMI and acute and chronic myocardial injury—which account for nearly half of all troponin elevations⁵—present a 1-year cardiovascular risk comparable to that of type 1 AMI, yet they systematically receive less investigation and cardioprotective treatment.^{4,5} This disparity is aggravated by the absence of specific therapeutic protocols for these

categories and by the predominance of noncardiovascular deaths—particularly infections and pulmonary decompensations—among patients with acute myocardial injury⁵, requiring an integrated clinical approach that extends beyond conventional ACS management.

The finding by Stelzle et al.⁴ that hs-cTnI predicts heart failure even within the normal reference range further expands the prognostic dimension of the biomarker, suggesting that patients with detectable troponin—particularly those in the upper quartiles of the reference range—may benefit from additional structural cardiac investigation, such as echocardiography and NT-proBNP measurement, strategies that are not currently routine in this group.⁴

Early CTCA, in turn, should be interpreted within its actual clinical scope: it is a tool with predominantly operational value, capable of reducing unnecessary invasive procedures and increasing diagnostic certainty, but without demonstrated prognostic benefit in intermediate-risk patients³. Its selective use—in scenarios of diagnostic uncertainty, in hospitals with limited access to invasive angiography, or in patients with elevated troponin without clear evidence of coronary obstruction—appears to be the most rational approach based on the available evidence.³ Comparison with studies of stable chest pain, in which CTCA demonstrated a reduction in long-term events through intensification of preventive therapy, reinforces that the impact of the test is strongly dependent on the pretest risk profile, the prevalence of obstructive disease, and the quality of the available standard care.³

Finally, the findings of this review point to the urgent need for randomized clinical trials evaluating specific secondary prevention strategies for type 2 AMI and myocardial injury,^{4,5} as well as studies defining the role of additional biomarkers and imaging methods in the prognostic stratification of patients with elevated hs-cTn without criteria for type 1 AMI.⁴ The integration of validated clinical scores, high-sensitivity biomarkers, and, when indicated, coronary imaging methods into structured diagnostic algorithms represents the most promising path toward improving outcomes in ACS.^{1,2,3,4,5}

CONCLUSION

Based on this study, optimizing risk stratification for patients with suspected acute coronary syndrome in the emergency setting depends on the synergistic integration of high-sensitivity biomarkers and validated clinical scores. The analyzed data confirm the superiority of the HEART score over traditional tools such as TIMI and GRACE, showing significantly greater discriminatory ability for predicting short-term major adverse cardiac events. While the HEART score was able to safely identify approximately 40% of the sample as low risk—with sensitivity above 95%, a negative predictive value of up to 99%, and minimal residual event rates—the traditional scores proved less efficient or overly conservative, limiting their usefulness in initial triage. This superior performance results from the alignment of the HEART score with the clinical reality of undifferentiated chest pain, allowing early identification of candidates for safe hospital discharge and reducing reliance on routine noninvasive testing after myocardial infarction has been ruled out using high-sensitivity troponin algorithms.

At the same time, the implementation of high-sensitivity troponins revealed a scenario of diagnostic progress accompanied by persistent therapeutic challenges. A substantial increase was observed in the recognition of type 2 myocardial infarction and acute and chronic myocardial injury, categories that now account for nearly half of troponin elevations in clinical practice. However, the findings highlight a critical gap: this increase in diagnosis was not accompanied by improved clinical outcomes at one year, demonstrating the absence of specific treatment protocols for non-atherosclerotic causes.

Patients with myocardial injury have cardiovascular risks comparable to those with type 1 myocardial

infarction, but systematically receive fewer protective interventions, indicating that effective detection is only the first step toward reducing morbidity and mortality. In addition, the ability of troponin to predict the risk of heart failure even at levels considered normal expands its prognostic value beyond acute diagnosis, suggesting that detectable concentrations may indicate the need for structured preventive monitoring. Regarding the role of coronary computed tomography angiography, its usefulness is essentially operational, as it is effective in avoiding unnecessary invasive procedures in intermediate-risk patients, although without a direct impact on mortality. Therefore, the evolution of care in acute coronary syndrome requires not only analytical and clinical precision in triage, but also the urgent development of clinical trials to define effective secondary prevention strategies for the new high-risk populations identified through high-sensitivity testing.

REFERENCES

1. O'Rielly CM, Harrison TG, Andruchow JE, Ronksley PE, Sajobi T, Robertson HL, Lorenzetti D, McRae AD. Risk scores for clinical risk stratification of emergency department patients with chest pain but no acute myocardial infarction: a systematic review. *Can J Cardiol.* 2023;39(3):304-310. doi: 10.1016/j.cjca.2022.12.028.
2. Poldervaart JM, Langedijk M, Backus BE, Dekker IMC, Six AJ, Doevendans PA, et al. Comparison of the GRACE, HEART and TIMI score to predict major adverse cardiac events in chest pain patients at the emergency department. *Int J Cardiol.* 2017;227:656-661. doi: 10.1016/j.ijcard.2016.10.080.
3. Gray AJ, Roobottom C, Smith JE, Goodacre S, Oatey K, O'Brien R, et al. Early computed tomography coronary angiography in patients with suspected acute coronary syndrome: randomised controlled trial. *BMJ.* 2021;374. doi: 10.1136/bmj.n2106.
4. Stelzle D, Shah ASV, Anand A, Strachan FE, Chapman AR, Denvir MA, et al. High-sensitivity cardiac troponin I and risk of heart failure in patients with suspected acute coronary syndrome: a cohort study. *Eur Heart J Qual Care Clin Outcomes.* 2018;4(1):36-42. doi: 10.1093/ehjqcco/qcx022.
5. Chapman AR, Adamson PD, Shah ASV, Anand A, Strachan FE, Ferry AV, Lee KK, Berry C, Findlay I, Cruikshank A, Reid A, Gray A, Collinson PO, Apple F, McAllister DA, Maguire D, Fox KAA, Vallejos CA, Keerie C, Weir CJ, Newby DE, Mills NL. High-sensitivity cardiac troponin and the universal definition of myocardial infarction. *Circulation.* 2020;141(3):161-171. doi: 10.1161/CIRCULATIONAHA.119.042960.

MAILING ADDRESS

GABRIEL FAGUNDES MOREIRA

5ª Avenida, s/n, esq. com a Rua 235, no Setor Leste Universitário, Goiânia/GO, Brazil.

E-mail: gabriel_fagundes@discente.ufg.br

EDITORIAL AND REVIEW

Chief editors:

Waldemar Naves do Amaral - <http://lattes.cnpq.br/4092560599116579> - <https://orcid.org/0000-0002-0824-1138>

Nílzio Antônio da Silva - <http://lattes.cnpq.br/1780564621664455> - <https://orcid.org/0000-0002-6133-0498>

Authors:

Gabriel Fagundes Moreira - <http://lattes.cnpq.br/8151873904023838> - <https://orcid.org/0009-0006-1140-5929>

Paulo Vinícius Dias - <http://lattes.cnpq.br/9861707511720132> - <https://orcid.org/0009-0006-5887-0115>

Matheus Lúcio Fernandes Gomes - <http://lattes.cnpq.br/7888430199844601> - <https://orcid.org/0009-0001-3083-2188>

Igor Diniz Alves - <http://lattes.cnpq.br/0009-0001-3083-2188> - <https://orcid.org/0009-0002-0752-7414>

Airton Silva de Santana - <http://lattes.cnpq.br/5673605880251211> - <https://orcid.org/0009-0002-9052-2402>

Library Review: Izabella Goulart

Spell Check: Dario Alvares

Translation: Soledad Montalbetti

Received: 24/08/26. Accepted: 27/08/26. Published in: 16/10/2026.