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Innovation in the early diagnosis of cardiovascular diseases through clinical biomarkers and predictive models to optimise therapeutic decision-making

Innovación en el diagnóstico temprano de enfermedades cardiovasculares mediante biomarcadores clínicos y modelos predictivos para la optimización de decisiones terapéuticas

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ABSTRACT

Introduction: Cardiovascular diseases require early detection strategies that integrate clinical and biological information. Biomarkers and predictive models offer complementary perspectives, although their interpretation depends on the healthcare context and the validation of each tool.
Objective: To examine the contributions of clinical biomarkers and predictive models to early cardiovascular diagnosis and to the guidance of preventive and therapeutic decisions.
Methodology: A qualitative, descriptive, and interpretative documentary review was conducted of publications on troponins, natriuretic peptides, inflammation, metabolism, and risk prediction. The selection record reported 440 identified documents, 92 duplicates, 348 screened records, 125 assessed reports, and 20 included documents. PRISMA 2020 was used to present the selection flow, without treating it as a systematic review method. Information was organized through thematic categories and narrative comparison.
Results: The synthesis distinguished biomarkers of myocardial injury, cardiac stress, inflammation, and atherosclerotic susceptibility. Troponins require serial and contextual interpretation; natriuretic peptides complement the assessment of heart failure. Inflammatory and lipid indicators characterize additional risk mechanisms. Predictive models integrate clinical and biochemical variables, but their performance depends on calibration, external validation, and data quality.
Conclusions: Integrating biomarkers and models may enrich cardiovascular risk stratification without replacing clinical judgment. Diagnostic usefulness, prognostic prediction, and improvements in therapeutic decisions are distinct outcomes requiring specific evaluation. This synthesis offers a documentary interpretation and does not demonstrate causal clinical effectiveness.

Keywords: Cardiovascular diseases; clinical biomarkers; early diagnosis; predictive models; precision medicine

RESUMEN

Introducción: Las enfermedades cardiovasculares requieren estrategias de detección temprana que integren información clínica y biológica. Los biomarcadores y modelos predictivos aportan perspectivas complementarias, aunque su interpretación depende del contexto asistencial y de la validación de cada herramienta.
Objetivo: Examinar los aportes de los biomarcadores clínicos y los modelos predictivos al diagnóstico

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temprano cardiovascular y a la orientación de decisiones preventivas y terapéuticas.
Metodología: Se desarrolló una revisión documental cualitativa, descriptiva e interpretativa de publicaciones sobre troponinas, péptidos natriuréticos, inflamación, metabolismo y predicción del riesgo. El registro de selección consignó 440 documentos identificados, 92 duplicados, 348 cribados, 125 evaluados y 20 incluidos. Se utilizó PRISMA 2020 como guía de presentación del flujo, sin equipararla con un método de revisión sistemática. La información se organizó mediante categorías temáticas y comparación narrativa.
Resultados: La síntesis distinguió biomarcadores de lesión miocárdica, estrés cardíaco, inflamación y susceptibilidad ateroesclerótica. Las troponinas requieren interpretación seriada y contextual; los péptidos natriuréticos complementan la evaluación de insuficiencia cardíaca. Los indicadores inflamatorios y lipídicos caracterizan mecanismos adicionales de riesgo. Los modelos predictivos integran variables clínicas y bioquímicas, pero su rendimiento depende de calibración, validación externa y calidad de datos.
Conclusiones: La integración de biomarcadores y modelos puede enriquecer la estratificación cardiovascular, sin reemplazar el juicio clínico. La utilidad diagnóstica, la predicción pronóstica y la mejora de decisiones terapéuticas constituyen resultados diferentes que requieren evaluación. La síntesis ofrece interpretación y no demuestra eficacia clínica causal.

Palabras clave: Enfermedades cardiovasculares; biomarcadores clínicos; diagnóstico temprano; modelos predictivos; medicina de precisión

INTRODUCTION

Cardiovascular diseases are among the leading causes of morbidity and mortality worldwide because of their complex aetiopathogenesis, progressive course and effects on organ function and survival. Their onset is linked to pathophysiological mechanisms encompassing atherosclerotic lesion formation, impaired endothelial function, activation of inflammatory responses, disturbances in lipid and glucose metabolism, and anatomical and functional changes in the heart and blood vessels. One distinctive feature of these conditions is that certain abnormalities may develop silently before producing recognisable clinical manifestations, making identification during the initial phases difficult. This situation highlights the need to strengthen detection strategies through procedures capable of recognising incipient physiological and biochemical changes, establishing individual susceptibility profiles and anticipating possible complications before they cause more severe cardiovascular damage.

The development of new diagnostic tools has encouraged the incorporation of clinical biomarkers and predictive systems into comprehensive cardiovascular risk assessment. Indicators such as high-sensitivity cardiac troponins, natriuretic peptides, high-sensitivity C-reactive protein and lipoprotein(a) help characterise different processes associated with myocardial injury, cardiac overload, inflammatory activity and atherothrombotic predisposition. These measurements are complemented by statistical algorithms and machine learning procedures whose processing capabilities facilitate the identification of associations between biochemical variables, medical history, haemodynamic parameters and individual characteristics. Combining these resources enables progress towards more precise assessment procedures, in which biological and computational information helps refine risk stratification, anticipate cardiovascular events and support the selection of therapeutic interventions appropriate to each patient's particular circumstances.

Detecting abnormal concentrations does not necessarily imply the presence of a specific disease: troponin indicates myocardial injury, but its interpretation requires symptoms, an electrocardiogram and serial changes (1). Similarly, natriuretic peptides complement the assessment of dyspnoea and possible heart failure (2). Lipoprotein(a), in turn, helps identify inherited atherosclerotic susceptibility (3). This distinction is crucial because a marker's performance depends on the population examined, the timing of measurement, disease prevalence and the purpose of the test. Research must also distinguish diagnostic accuracy from prognostic capability and therapeutic benefit: a marker may be associated with an outcome without demonstrating that its incorporation changes clinical decisions or reduces adverse events. Each result must therefore be interpreted by relating the test to the specific clinical suspicion and the purpose of the assessment, avoiding conclusions based on an isolated indicator.

Recognising cardiovascular diseases in their early phases remains constrained by diagnostic limitations related to heterogeneous clinical presentations, the coexistence of predisposing factors and the ability of conventional procedures to detect subclinical abnormalities. Certain conditions progress without specific symptoms, so recognition may occur after organ damage or complications such as myocardial infarction, heart failure and stroke have developed. This situation highlights the need to incorporate biochemical and physiological indicators that identify pathological processes at early stages and complement information obtained through clinical examination and conventional investigations. Traditional stratification systems also vary in discrimination and calibration, particularly in populations with different demographic, metabolic, genetic and epidemiological characteristics, potentially reducing the accuracy of individual risk estimates.

Integrating and interpreting information from different diagnostic sources is a significant limitation because laboratory results, medical history, haemodynamic parameters and clinical records require joint assessment to establish reliable estimates. Although biomarkers and predictive models offer alternatives for improving identification of patients with high cardiovascular susceptibility, their incorporation requires demonstration of analytical validity, discrimination, reproducibility and usefulness in medical decision-making. Using insufficiently validated algorithms may cause misclassification, inaccurate estimates and inappropriate therapeutic recommendations, particularly when systems have limited generalisability or incorporate poorly representative data. Joint assessment must preserve a distinction between the information provided by each tool and the decisions that can actually be supported by that information.

Differences in technological infrastructure, availability of specialised tests and the operational capacity of health services must also be considered. It is therefore relevant to examine how clinical biomarkers and predictive models contribute to early recognition of cardiovascular diseases and their potential to improve diagnostic accuracy and guide individualised therapeutic interventions based on safety and clinical appropriateness. The general objective was to examine the contributions of clinical biomarkers and predictive models to early cardiovascular diagnosis and the guidance of therapeutic decisions. The research question was: how do these resources contribute to identifying abnormalities, estimating risk and making clinical decisions? Within this framework, the review

maintains an interpretative perspective on the available evidence and distinguishes potential applications from demonstrated clinical outcomes.

LITERATURE REVIEW

Evolution of cardiovascular stratification

The development of preventive cardiology has been accompanied by assessment systems designed to recognise factors associated with the onset of cardiovascular diseases and estimate the probability of adverse clinical events. Early multivariable stratification procedures integrated parameters such as age, blood pressure, cholesterol concentrations, smoking and metabolic history, enabling individual estimates of cardiovascular susceptibility. The Framingham study is a prominent contribution: its findings established relationships between modifiable risk factors and the onset of coronary disease. The historical analysis of cardiovascular risk described the evolution of the cardiovascular risk concept by characterising factors associated with coronary disease and their application in preventive assessment (4). Prediction equations subsequently incorporated more sophisticated statistical procedures and different population characteristics, although limitations in individual accuracy encouraged the incorporation of complementary biological indicators and computational tools.

An analysis of risk models described the construction of cardiovascular prediction models and their implications for risk classification (5). Its contribution helps distinguish identification of associated factors from estimation of individual probabilities. In this study's analysis, that difference is crucial: the presence of hypertension or dyslipidaemia is not equivalent to a sufficiently calibrated prediction. The analysis of hypertension control examined persistent difficulties in controlling arterial hypertension, a condition linking statistical stratification to practical prevention possibilities (6). Risk assessment therefore requires interpretation of both the estimated probability and the availability of effective interventions and clinical follow-up. This evolution shows that stratification is not an invariable measurement but an estimate conditioned by the population and the variables incorporated.

Incorporating biomarkers into diagnosis

The use of circulating biomarkers changed cardiovascular assessment procedures by providing biochemical information about abnormalities that cannot always be recognised through physical examination or conventional clinical parameters. Cardiac troponins became particularly important as indicators of myocardial injury, while natriuretic peptides expanded the possibilities for assessing wall stress and haemodynamic disturbances. The review on troponin interpretation analysed clinical criteria for interpreting cardiac troponin concentrations and their relationship to myocardial infarction diagnosis, distinguishing myocardial injury from other clinical conditions (1). Subsequently, high-sensitivity assays enabled detection of low concentrations of these proteins and refined evaluation algorithms for patients with suspected acute coronary syndrome.

In this field, contemporary clinical recommendations emphasise serial measurements and temporal changes in concentrations to improve diagnostic accuracy. The study examined analytical characteristics of troponin measurement methods and their diagnostic possibilities (7). Assay

sensitivity improves detection of low concentrations but also requires the biochemical finding to be distinguished from the aetiological diagnosis. The clinical review addressed the clinical usefulness of cardiac biomarkers in different cardiovascular diseases (8). Comparing these studies shows that a test's performance depends on its specific purpose: recognising myocardial injury, contributing to a differential diagnosis or estimating prognosis. This distinction prevents results obtained using markers representing different pathophysiological processes from being interpreted as equivalent. These markers' contributions must be examined in relation to the healthcare setting and the complementary tests available for diagnosis.

Inflammation and atherosclerotic progression

Studying inflammatory mechanisms broadened understanding of the biological processes involved in cardiovascular disease progression, particularly their participation in endothelial dysfunction and atherosclerotic lesion formation. High-sensitivity C-reactive protein became relevant as an indicator of systemic inflammation and a possible complement to traditional cardiovascular stratification parameters. The analysis of atherosclerotic inflammation highlighted this marker's importance in coronary risk assessment, noting its association with inflammatory processes linked to atherosclerosis (9). Immunological mediators, including interleukin-6, fibrinogen and other proinflammatory cytokines, also helped characterise mechanisms associated with plaque instability and atherothrombotic complications. These contributions broadened cardiovascular risk interpretation by incorporating inflammatory and metabolic variables that complement lipid profiles and haemodynamic indicators.

From a pathophysiological perspective, the research explained atherosclerosis as a dynamic process involving interactions among lipids, immunity and vascular wall changes (10). This interpretation situates inflammation within a pathophysiological sequence rather than treating it as an independent indicator of disease. The study reviewed inflammatory mechanisms and therapeutic alternatives related to atherosclerosis (11). Both approaches converge in recognising the importance of inflammatory pathways; they differ in the level of evidence required to translate a biological association into a therapeutic decision. An isolated elevation of C-reactive protein cannot specifically attribute inflammation to a coronary plaque. Systemic inflammation must be interpreted cautiously because it cannot, by itself, locate a particular atherosclerotic lesion. This distinction helps prevent the plausibility of a pathophysiological mechanism from being confused with sufficient evidence to adopt a specific intervention.

Natriuretic peptides and heart failure

Biomarkers associated with cardiac function have become particularly important in identifying abnormalities related to myocardial stress and heart failure. These indicators include B-type natriuretic peptide (BNP) and its N-terminal fragment (NT-proBNP), whose concentrations provide information about loading conditions and distension of the cardiac chambers. The BNP review examined BNP's role in evaluating acute dyspnoea in patients with dyspnoea, helping distinguish possible cardiac and non-cardiac causes of this clinical manifestation (2). Its incorporation into assessment procedures facilitates interpretation of symptoms such as breathlessness, exercise

intolerance and fluid retention, particularly when complemented by echocardiography and functional assessment. These markers also provide prognostic information and identify patients requiring specialised cardiovascular follow-up.

Interpreting BNP and NT-proBNP requires consideration of age, renal function and clinical conditions that modify their concentrations. In acute dyspnoea assessment, these indicators help organise diagnostic hypotheses but do not replace history-taking, physical examination or imaging. They are more useful when the measured value is integrated with the pre-test clinical probability of heart failure. This interpretation also avoids confusing an elevation consistent with haemodynamic overload with confirmation of a single cause. Diagnostic reasoning requires establishing which question each marker answers and what uncertainty remains after the result is known. The laboratory result therefore becomes meaningful when interpreted alongside symptoms, physical examination and other cardiac assessment tests.

Lipoprotein(a) and hereditary risk

Lipoprotein(a) has become an important biomarker in atherosclerotic risk assessment because of its relationship with thrombotic mechanisms, vascular abnormalities and cardiovascular complications of hereditary origin. Its plasma concentration is strongly genetically determined and provides information complementary to LDL cholesterol and other lipid profile components. The study on lipoprotein(a) analysed its clinical relevance and links to atherosclerotic risk through which this lipoprotein component participates in atherosclerotic progression and cardiovascular events (3). Its measurement identifies susceptibility profiles that conventional lipid parameters might miss, particularly in people with a family history of premature cardiovascular disease. Incorporation into clinical assessment therefore supports a more complete characterisation of risk and helps establish preventive measures tailored to individual circumstances.

From a hereditary perspective, lipoprotein(a) concentration provides an inherited dimension that routine LDL cholesterol measurement does not fully represent. The LDL cholesterol analysis examined (12) evidence for the causal relationship between LDL and atherosclerotic cardiovascular disease, whereas the publication on lipoprotein(a) (3) focused on risk associated with lipoprotein(a). The comparison distinguishes two related but non-interchangeable lipid determinants. Their integration is particularly relevant when there is a family history of premature disease or disagreement between conventional risk estimates and clinical history. Preventive decisions must consider exposure magnitude and individual context. This measurement's preventive value depends on interpretation alongside established risk factors and personal and family history.

Machine learning and clinical data

Artificial intelligence and machine learning have expanded the possibilities for processing clinical information through algorithms capable of recognising complex relationships among biochemical variables, medical history and physiological parameters. These tools analyse electronic health records, laboratory results, electrocardiograms and demographic characteristics to estimate cardiovascular event probabilities. The review on precision cardiovascular medicine addressed possibilities for machine learning in cardiovascular risk prediction using electronic health records,

highlighting its capacity to integrate heterogeneous information and recognise patterns that may be difficult to identify through traditional statistical procedures (13). However, application requires consideration of data quality, the representativeness of training populations and external algorithm validation.

These criteria are essential to ensure that generated estimates retain adequate accuracy and can be safely transferred to care procedures. The genetics and artificial intelligence study addressed integration of genetics and artificial intelligence to estimate cardiovascular risk, expanding the variables available for prediction (14). The umbrella review on cardiovascular artificial intelligence examined AI applications and difficulties in transferring models into clinical practice (15). Both approaches distinguish pattern recognition capabilities from demonstrated clinical benefit. Training on heterogeneous records may improve apparent discrimination, but generalisation requires testing performance across different institutions, periods and populations. Without external validation, estimates do not, by themselves, justify therapeutic changes.

Integrating biomarkers and multivariable models

Combining circulating biomarkers with multivariable models has become important in the search for more precise procedures to classify cardiovascular susceptibility and recognise patients who might benefit from specific preventive interventions. The scientific statement on model assessment examined the contribution of different biomarkers to cardiovascular event prediction, emphasising the importance of assessing their ability to provide information beyond conventional risk factors (16). Within this perspective, cardiac troponins, natriuretic peptides and inflammatory indicators can complement traditional estimates by identifying biological abnormalities associated with different pathophysiological mechanisms. Integration can refine classification of certain patients, especially those in intermediate risk categories. Nevertheless, clinical use requires demonstration that the additional information produces relevant changes in preventive and therapeutic decisions.

Adding further variables may increase an equation's complexity without improving its usefulness. The review of risk models provides a framework for interpreting modelling decisions, while the scientific statement on model assessment proposes (16) evaluating the incremental usefulness of new biomarkers and tools. The comparison shows that a change in area under the curve does not necessarily equate to reclassification relevant to the patient. It is also important to determine whether the model identifies people who would receive a different intervention and whether that change produces a favourable balance of benefits and harms. Prediction and clinical decisions therefore require related but distinct assessments. The value of integration must be judged by the information it adds and its ability to guide appropriate decisions, not solely by model complexity.

Validation and clinical usefulness of models

Contemporary criteria for evaluating biomarkers and predictive systems include requirements for statistical performance, reproducibility and the clinical usefulness of diagnostic tools. The scientific statement on model assessment, through an American Heart Association scientific statement, emphasised the importance of assessing the accuracy, calibration, discrimination, incremental contribution and applicability of new resources for cardiovascular risk estimation (16). This

perspective encompasses genomic, proteomic and metabolomic information and AI algorithms to characterise individual conditions more comprehensively. However, technological sophistication does not automatically guarantee greater predictive capability because results also depend on data quality, validation in independent populations and the interpretability of generated estimates.

These tools must therefore be incorporated on the basis of equity, reproducibility and demonstrable clinical benefit. Artificial intelligence models require particular scrutiny when incorporating genetic variables, biomarkers with unequal availability or incomplete clinical records. The umbrella review on cardiovascular artificial intelligence identifies translational difficulties that strong internal performance does not resolve (15). These include prevalence variation, coding differences and limited interpretability of some estimates. Validation must include calibration, discrimination and assessment of impact on actual decisions. A tool may maintain acceptable classification while overestimating absolute risk in a population different from the development population. Comprehensive assessment also requires attention to variable availability and the possibility of applying the tool consistently.

Pathophysiological mechanisms of cardiovascular diseases

Cardiovascular diseases arise through interrelated pathophysiological processes that progressively compromise vascular integrity, tissue perfusion and cardiac muscle function. Atherosclerosis is a prominent mechanism, characterised by lipoprotein particle retention in the arterial intima, inflammatory cell activation and lesions that alter blood vessel architecture. Endothelial dysfunction also contributes by reducing the availability of vasodilatory mechanisms and modifying the endothelium's anticoagulant and anti-inflammatory properties. The analysis of atherosclerotic inflammation explained that atherosclerotic progression involves interactions among immune cells, modified lipids and arterial wall components, whose activity conditions plaque development and vulnerability (9). These mechanisms support incorporation of biochemical indicators capable of identifying inflammatory, metabolic and myocardial abnormalities before clinically recognisable cardiovascular complications emerge.

Understanding atherosclerosis from a contemporary perspective requires integration of lipid accumulation mechanisms and local immune responses. The research described how this interaction changes vascular lesion progression (10). Diagnostically, a sustained inflammatory process does not justify using a non-specific biomarker as the sole test for coronary disease. Interpretation requires plausible relationships among the biological mechanism, the test used and the outcome of interest. This reasoning avoids excessive inferences when an epidemiological association is presented as demonstrating the presence or severity of a specific lesion. The relationship among these processes explains why biological markers provide complementary information while clinical confirmation remains necessary. Coherence among mechanism, measurement and decision is essential for interpreting the evidence assembled in this review appropriately.

Clinical biomarkers and applications in cardiovascular diagnosis

Clinical biomarkers are measurable biological parameters that characterise physiological processes, identify pathological abnormalities and assess possible changes associated with disease progression. Their measurement through analytical procedures provides information complementary to physical

examination, electrocardiography and imaging techniques used in cardiology. Widely used indicators include cardiac troponins, natriuretic peptides, high-sensitivity C-reactive protein, lipoprotein(a) and various metabolic parameters. The publication on clinical establishment of biomarkers examined the conditions necessary to incorporate biomarkers into clinical practice, considering their ability to provide information on specific biological processes and contribute to identifying pathological conditions (17). Their diagnostic performance depends on properties such as sensitivity, specificity, analytical precision and reference values, as well as correspondence between the marker and the pathophysiological mechanism assessed.

Interpreting these parameters jointly broadens characterisation of cardiovascular status and reduces reliance on individual measurements. A biomarker's clinical usefulness also depends on measurement reproducibility and the decision it aims to guide. The clinical review (8) described applications of cardiac markers in cardiovascular diseases; the publication on clinical establishment of biomarkers analysed (17) conditions for incorporation into practice. The comparison places a test's value at three levels: analytical accuracy, association with a clinical condition and ability to improve a decision. A marker may fulfil the first two requirements while still lacking sufficient evidence for the third. Thresholds and result interpretation must therefore be adapted to the application context.

High-sensitivity cardiac troponins and detection of myocardial injury

Cardiac troponins I and T are proteins in the cardiomyocyte contractile apparatus and play a fundamental role in regulating muscle contraction. Their presence in the circulation indicates myocardial injury because their release increases when abnormalities compromise cardiac cell integrity. High-sensitivity assays quantify low concentrations and recognise variations that may be relevant during early phases of certain cardiovascular events. The review on troponin interpretation examined criteria for interpreting troponin concentrations and distinguishing myocardial injury from other conditions, considering rising or falling changes and clinical manifestations consistent with ischaemia (1). An elevated concentration therefore does not independently confirm myocardial infarction, since it may also occur in heart failure, myocarditis, renal disease and other systemic disturbances.

Interpretation requires integrating laboratory results with the electrocardiogram, clinical assessment and complementary diagnostic procedures. Recognising myocardial injury requires distinguishing troponin elevation from clinical evidence of ischaemia. The study (7) highlighted analytical features of available assays, while the troponin interpretation review examined (1) interpretation in the high-sensitivity era. Both support an approach based on concentrations, temporal changes and clinical context. Elevation may accompany processes other than acute infarction, including haemodynamic disorders and renal disease. Decisions about further tests and treatment must therefore depend on integrated clinical and electrocardiographic information rather than an isolated value. This distinction must be maintained even when high-sensitivity analytical methods are used in emergency care settings.

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Inflammatory biomarkers and vascular pathophysiology

In the vascular system, inflammation contributes to different stages of cardiovascular disease through mechanisms affecting endothelial activity, immune responses and atherosclerotic lesion stability. High-sensitivity C-reactive protein is used to characterise systemic inflammation and complement cardiovascular risk assessment, particularly in people with predisposing factors. Inflammatory mechanisms contribute to atherosclerotic progression and to understanding cardiovascular events, particularly through the relationship between immune responses and vascular lesion progression (9). Mediators such as interleukin-6, fibrinogen and other cytokines involved in inflammatory signalling pathways are also important and may promote conditions related to atherothrombosis. Measuring these indicators broadens biological risk characterisation, although concentrations must be interpreted with consideration of possible infections, autoimmune processes and other diseases capable of modifying the inflammatory response.

As a systemic indicator, C-reactive protein reflects a systemic inflammatory response and lacks specificity for locating a vascular lesion. The study examined changes in C-reactive protein and other cellular markers during periodontal treatment, illustrating the influence of extracardiac inflammatory processes (18). This contextual finding requires caution in cardiovascular estimation. The analysis of atherosclerotic inflammation places inflammation within atherosclerotic pathophysiology, but this does not mean that every marker elevation identifies plaque activity (9). Biological association must be distinguished from a test's specific diagnostic capability. The limited specificity of inflammatory indicators requires consideration of alternative causes of elevation before attributing them to vascular disease. This comparison reinforces the need to distinguish the pathophysiological mechanism studied from a test's ability to identify it in a particular patient.

Metabolic biomarkers and atherosclerotic susceptibility

Disturbances in lipid and glucose metabolism are relevant mechanisms in cardiovascular disease onset and progression because of their relationship with lipid accumulation in arterial walls, endothelial dysfunction and impaired metabolic regulation. Low-density lipoprotein (LDL) cholesterol, triglycerides, glycated haemoglobin and lipoprotein(a) characterise different conditions associated with atherosclerotic lesion development. The LDL cholesterol research analysed the causal role of LDL cholesterol concentrations in atherosclerotic cardiovascular disease, emphasising cumulative exposure to these particles throughout life (12). Lipoprotein(a) is also of particular interest because of its predominantly genetic determination and its association with cardiovascular events and aortic valve stenosis.

Assessed jointly, these indicators identify potentially asymptomatic metabolic abnormalities and support preventive strategies for lipid control, glucose regulation and vascular risk reduction. Metabolic risk depends on cumulative exposure to modifiable and hereditary factors as well as a single measurement. The LDL cholesterol analysis examined (12) the causal importance of LDL cholesterol in atherosclerotic disease, while the hypertension control analysis addressed (6) difficulties in controlling hypertension. These studies interpret risk as the result of distinct mechanisms that may coexist and amplify one another. Prevention therefore requires simultaneous

assessment of lipids, blood pressure, history and conditions affecting therapeutic adherence. New biomarkers must justify what additional information they provide beyond those established factors.

Predictive models and cardiovascular risk estimation

Predictive models are statistical and mathematical instruments that estimate the probability of clinical events by processing individual variables together. In cardiology, they incorporate information on age, blood pressure, lipid concentrations, smoking, diabetes mellitus and medical history, among other relevant parameters. Their application establishes risk categories and guides the intensity of preventive measures according to each patient's characteristics. The scientific statement on model assessment established criteria for evaluating new models, biomarkers and tools without presenting a prediction equation developed by its authors, contributing to broader possibilities for individual estimation in different populations (16). More recent systems also incorporate circulating biomarkers and additional clinical parameters to improve discrimination among individuals with different probabilities of cardiovascular events.

Their use requires assessment of calibration, sensitivity, specificity, discrimination and external validation because a tool's performance may vary according to the demographic, epidemiological and clinical characteristics of the population where it is applied. Cardiovascular prediction must distinguish statistical performance from the ability to change clinical behaviour. The risk model review analysed (5) risk models and their foundations, while the scientific statement on model assessment established (16) criteria for judging new tools. A model contributes more than ranking patients by probability: it must also offer sufficiently calibrated estimates to support intervention thresholds. When an algorithm is applied in different populations, risk factor distributions and event incidence may alter performance. External validation is essential before translating its results into preventive decisions.

Artificial intelligence and machine learning applied to cardiology

Artificial intelligence encompasses computational systems designed to process information, identify relationships among variables and generate results through automated analytical procedures. One principal application is machine learning, which uses algorithms trained on datasets to recognise patterns and establish estimates from heterogeneous clinical information. In cardiology, these tools integrate laboratory results, electrocardiograms, diagnostic images, physiological records and electronic health records to identify associations related to cardiovascular diseases. The precision cardiovascular medicine review described the potential of artificial intelligence in cardiovascular medicine, highlighting applications in clinical event prediction, image interpretation and diagnostic pattern recognition (13). Classification, regression and deep learning algorithms can complement conventional procedures by analysing numerous variables simultaneously.

However, incorporation requires representative datasets, validation in independent populations, algorithmic transparency and professional oversight to reduce errors arising from bias, incomplete information or insufficiently generalisable estimates. The AI-supported syncope case presented probably arrhythmic syncope incorporating a dialogue with artificial intelligence (19). Its contribution illustrates potential support for reasoning, but a clinical case does not, by itself, establish

generalisable diagnostic accuracy. The Takotsubo case described Takotsubo cardiomyopathy in an oncological context, reminding readers that cardiovascular manifestations may depend on complex clinical circumstances (20). Both studies demonstrate the importance of retaining differential judgement when considering automated recommendations. Technology can organise hypotheses, but diagnostic confirmation requires appropriate tests and individual assessment.

Precision medicine and optimisation of therapeutic decisions

In cardiology, precision medicine aims to adapt preventive, diagnostic and therapeutic strategies to each individual's biological, clinical and genetic characteristics, moving beyond procedures based exclusively on general population criteria. Its application in cardiology is strengthened by integrating biomarkers and predictive models that characterise susceptibility profiles and estimate the possible course of cardiovascular abnormalities. The precision cardiovascular medicine review highlighted the importance of healthcare based on individual characteristics, integrating biological and technological information to improve intervention selection (13). This perspective guides decisions concerning lipid control, hypertension treatment, prevention of thrombotic events and selection of pharmacological interventions according to identified clinical conditions.

Ensuring therapeutic appropriateness requires algorithmic results to be interpreted alongside medical judgement, current clinical guidelines, contraindications and patient preferences. Integrating advanced diagnostic tools can thus help improve the timeliness, safety and appropriateness of cardiovascular interventions. Precision cardiology integrates genetic, clinical and biochemical data to tailor prevention and treatment to individual characteristics. The genetics and artificial intelligence study examined (14) their combination, while the precision cardiovascular medicine review (13) broadened the approach through pharmacogenomics and real-world evidence. The comparison reveals an opportunity for personalisation while also raising validation and equity requirements. Unequal availability of genetic tests or algorithms may limit application of these strategies. Therapeutic selection must depend on verifiable clinical benefits not exclusively on the sophistication of the processed information.

METHOD

The study used a qualitative approach with a descriptive and interpretative documentary design to analyse the contributions of clinical biomarkers and predictive models to early diagnosis of cardiovascular diseases. The research procedure consisted of a structured documentary search, selection by relevance and thematic synthesis of scientific publications. The PRISMA 2020 statement was used exclusively to guide presentation of the identification, screening, eligibility and inclusion flow; it was not the analytical method, and the design was not assumed to constitute a systematic review. Information was collected by consulting six academic sources: Scopus, SciELO, Relacyt, Dialnet, Latindex and Google Scholar. Publications from 2016-2026 were considered, with emphasis on documents concerning early detection of cardiovascular diseases, clinical biomarkers, inflammatory and metabolic indicators, predictive algorithms, machine learning and cardiovascular risk stratification.

To retrieve relevant literature, the strategy combined Spanish and English terms using Boolean operators to broaden document retrieval and refine thematic correspondence. Combinations included expressions such as: enfermedades cardiovasculares AND diagnóstico temprano, biomarcadores clínicos AND riesgo cardiovascular, cardiovascular biomarkers AND early diagnosis, predictive models AND cardiovascular disease and machine learning AND cardiovascular risk. Combinations related to cardiac troponins, natriuretic peptides, C-reactive protein, lipoprotein(a), artificial intelligence and cardiovascular event prediction were also used. Selection criteria were established to ensure relevance to the topic. Scientific articles published between 2016 and 2026, full-text documents, Spanish or English publications, and studies analysing clinical biomarkers, early detection procedures, predictive tools or applications related to cardiovascular risk estimation were included. Documents providing information about the diagnostic usefulness of biochemical indicators and their relationship with therapeutic decisions were also considered.

Duplicate publications, documents without full-text access, studies not directly related to cardiovascular diseases, studies focused exclusively on non-cardiovascular conditions and publications addressing neither biomarkers nor predictive procedures were excluded. Records with insufficient information to answer the research question and documents whose thematic orientation did not match the established criteria were also discarded. The initial search identified 440 records from the six sources: 96 from Scopus, 74 from SciELO, 58 from Relacyt, 62 from Dialnet, 55 from Latindex and 95 from Google Scholar. Initial cleaning identified 92 duplicate records, which were removed before thematic assessment. Titles and abstracts of the remaining 348 documents were examined, resulting in exclusion of 218 records that did not meet the established criteria.

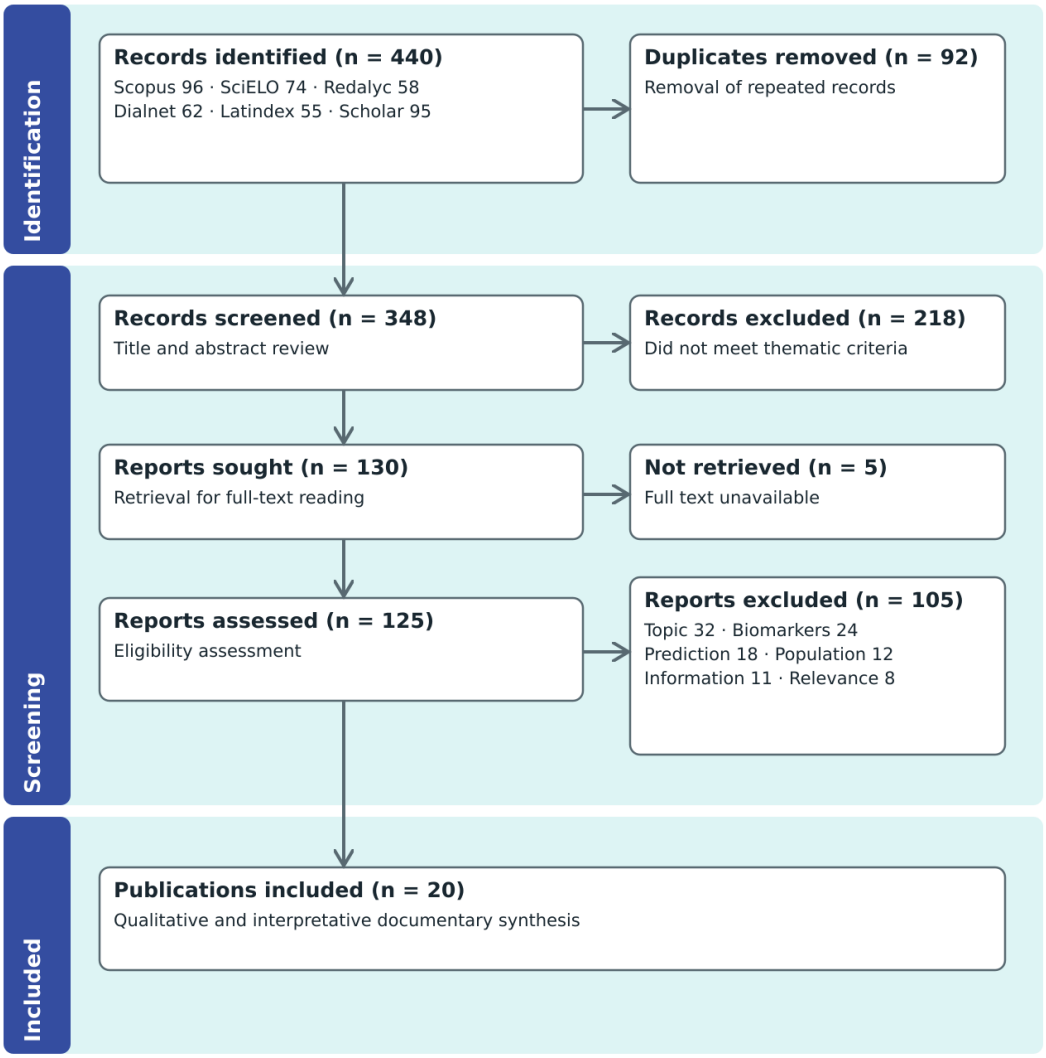
Literature retrieval began in September 2026. The described thematic combinations and selection criteria were applied, with deduplication before screening and subsequent full-text assessment. The selection flow is presented through counts for each stage. Retrieval of 130 documents for comprehensive assessment was then attempted; however, 5 could not be obtained in full text, leaving 125 publications for eligibility assessment. At this stage, 105 documents were excluded for different reasons: 32 did not directly address early diagnosis of cardiovascular diseases; 24 did not analyse clinical biomarkers; 18 incorporated neither predictive models nor risk estimation procedures; 12 focused on unrelated populations or conditions; 11 provided insufficient information for analysis; and 8 did not fulfil currency or thematic relevance criteria.

This procedure produced a final corpus of 20 documents selected for correspondence with the study's central categories. Information from selected documents was organised in a documentary analysis matrix developed in Microsoft Excel. This tool recorded each publication's bibliographic details, year, origin, central topic, biomarkers examined, diagnostic procedures, predictive model characteristics and principal clinical contributions. Classification was structured into four categories: clinical biomarkers for cardiovascular detection, indicators of myocardial injury and inflammation, predictive models for risk estimation, and contributions of these tools to therapeutic decision-making. Information was processed through qualitative content analysis, identifying similarities, differences and conceptual contributions among the selected documents.

This procedure enabled interpretation of the usefulness of biomarkers and predictive systems as complementary resources for recognising early cardiovascular abnormalities, estimating individual susceptibility and guiding informed clinical decisions. Document selection and organisation followed the identification, screening, eligibility and inclusion stages in PRISMA 2020. The chosen approach organised findings by conceptual and clinical contribution without attributing a quantitative effectiveness estimate to the documentary corpus. Sources were used to locate and consult relevant literature, taking account of differences among search engines, databases and academic directories. Search expressions targeted documents on the clinical and predictive components of the problem according to the planned analytical categories. Thematic relevance was assessed by considering the explicit relationship between each publication's objectives and the questions addressed in the documentary review.

Figure 1

Document selection flow diagram according to PRISMA 2020



Note. Data correspond to the figures reported in the methodology. Source: prepared by the author.

RESULTS

Clinical biomarkers made distinct contributions to early detection of cardiovascular abnormalities. High-sensitivity cardiac troponins identified myocardial injury through elevated concentrations and temporal variations; high-sensitivity C-reactive protein provided information on inflammatory processes related to atherosclerotic progression, while BNP and NT-proBNP helped identify abnormalities associated with cardiac chamber stress and distension. Lipoprotein(a) also identified atherosclerotic susceptibility linked to hereditary factors. LDL cholesterol, triglycerides and glycated haemoglobin provided information on metabolic abnormalities associated with vascular deterioration. For cardiovascular risk estimation, the examined publications described multivariable equations integrating age, blood pressure, lipid concentrations, smoking, diabetes and clinical history to estimate adverse event probabilities.

Biomarkers complemented these estimates with information on myocardial, inflammatory and metabolic abnormalities. Machine learning models and artificial intelligence tools likewise processed electronic health records, laboratory tests, electrocardiograms and imaging studies. Their application facilitated pattern recognition and individual risk classification, although performance depended on data quality, calibration and validation in independent populations. Regarding therapeutic decisions, integrating biomarkers and predictive models provided complementary information to guide preventive measures and clinical interventions. This combination characterised individual conditions, identified higher susceptibility profiles and supported actions concerning lipid control, blood pressure regulation and thrombotic event prevention.

Within precision medicine, these resources were incorporated to adapt interventions to each patient's biological and clinical characteristics. However, application required consideration of clinical guidelines, possible contraindications and professional judgement so that computational estimates complemented medical assessment. Comparing these indicators distinguished biological mechanisms and contexts of use rather than treating them as interchangeable tests for the same disease. Publications also emphasised checking model performance outside the development data. The tools' contribution was therefore interpreted as decision support rather than, by itself, a demonstration of therapeutic improvements.

Table 1

Matrix of the 20 included publications: identification, topic, design and contribution to the categories analysed.

Reference and author	Title / topic	Publication type	Finding and contribution to the synthesis
1. Maayah et al. (2024)	Clinical interpretation of high-sensitivity serum troponin	Clinical review	Distinguishes myocardial injury, infarction diagnosis and the need for clinical context.

Reference and author	Title / topic	Publication type	Finding and contribution to the synthesis
2. Morales and Rada (2019)	B-type natriuretic peptide in acute dyspnoea	Clinical evidence review	Examines BNP as an aid in assessing dyspnoea of possible cardiac origin.
3. Hameau et al. (2024)	Lipoprotein(a): underdiagnosed risk	Clinical review	Focuses on lipoprotein(a) as an atherosclerotic risk factor.
4. Cusumano and Victoria (2025)	Evolution of the cardiovascular and renal risk factor concept	Historical review	Contextualises the evolution of risk factors and cardiovascular prevention.
5. Donado et al. (2017)	Cardiovascular risk prediction models	Review	Describes construction, interpretation and validation of risk models.
6. Sosa et al. (2022)	Arterial hypertension control	Public health analysis	Identifies persistent difficulties in controlling a modifiable risk factor.
7. Chaulin (2021)	Cardiac troponins: analytical characteristics	Review	Examines analytical assays and limitations of diagnostic interpretation.
8. Escudero et al. (2025)	Clinical usefulness of cardiac biomarkers	Clinical review	Summarises biomarker applications in cardiovascular diseases.
9. Ait-Oufella and Libby (2024)	Inflammation and atherosclerosis: prospects for trials	Review of mechanisms and trials	Explains inflammatory pathways and their potential translation into interventions.
10. Libby (2021)	The changing landscape of atherosclerosis	Pathophysiological review	Relates lipids, immunity and vascular lesion progression.
11. Hidalgo (2025)	Inflammation in atherosclerosis: mechanisms and treatments	Clinical overview	Synthesises inflammatory mechanisms and therapeutic options.

Reference and author	Title / topic	Publication type	Finding and contribution to the synthesis
12. Corral and Lavalle (2024)	LDL cholesterol and atherosclerotic causality	Critical analysis	Discusses causal evidence for LDL and its preventive relevance.
13. Alshahrani et al. (2026)	Precision cardiovascular medicine	Review	Integrates pharmacogenomics, biomarkers, AI and real-world evidence.
14. Ceja et al. (2025)	Genetics and artificial intelligence in cardiovascular prediction	Predictive modelling study	Examines integration of polygenic risk, ECG and clinical information.
15. Parizad et al. (2026)	AI for cardiovascular risk prediction	Umbrella review	Identifies applications and barriers to validation and clinical translation.
16. Khan et al. (2026)	Criteria for evaluating models and biomarkers	Scientific statement	Defines discrimination, calibration, incremental usefulness and clinical benefit.
17. García and Carmona (2024)	Establishing biomarkers in clinical practice	Methodological analysis	Distinguishes biomarker properties and implementation conditions.
18. Rojas (2025)	Periodontal treatment and C-reactive protein	Clinical dental study	Illustrates the influence of extracardiac inflammation on systemic markers.
19. Cagide et al. (2026)	Arrhythmic syncope and artificial intelligence	Case report	Illustrates AI use in clinical reasoning; does not establish general effectiveness.
20. García et al. (2025)	Takotsubo in a patient with laryngeal carcinoma	Case report	Illustrates the complexity of cardiovascular differential diagnosis.

Note. Each row corresponds to a unique publication in the Vancouver bibliography. The synthesis distinguishes mechanisms, diagnostic tests, prediction models and case reports; results cannot be compared directly using a common metric. Source: prepared by the author.

Table 2

Comparative synthesis of findings and application limits

Dimension	Sources	Population or context	Finding and limitation
Troponins	(4,5,6)	Suspected myocardial injury	Serial and clinical interpretation is required; elevation is not equated with infarction.
Natriuretic peptides	(10)	Patients with acute dyspnoea	BNP supports differential diagnosis without replacing clinical assessment.
Inflammation and lipids	(7,8,11,12,18)	Atherosclerosis; hereditary risk; extracardiac inflammation	Complementary mechanisms; CRP does not identify coronary injury by itself.
Prediction and precision medicine	(2,13,14,15,16)	At-risk populations and heterogeneous clinical data	Calibration, external validation and incremental usefulness are distinct requirements.
Case reports	(19,20)	Individual patients	Illustrate diagnostic complexity without estimating sensitivity or general effectiveness.

Note. The dimensions, sources, populations or contexts and application limits of the included publications are compared. Source: prepared by the author.

The comparison distinguishes test results, pathophysiological mechanisms, model evaluations and case descriptions.

DISCUSSION

Incorporating clinical biomarkers into cardiovascular assessment represents a significant advance over procedures based exclusively on conventional risk factors. Although traditional equations estimate cardiovascular event probabilities using variables such as age, blood pressure, smoking and lipid concentrations, their ability to recognise individual biological abnormalities has certain limitations. In this respect, the scientific statement on model assessment distinguished discrimination, calibration and incremental clinical usefulness; its proposal requires demonstration that a biomarker changes decisions and does not merely improve a statistical indicator (16). This relationship shows that combining clinical and biochemical parameters broadens risk characterisation, particularly in patients whose susceptibility cannot be established solely through

conventional indicators. The diagnostic usefulness of biomarkers also depends on their correspondence with the pathophysiological mechanisms of each cardiovascular abnormality.

Cardiac troponins identify myocardial injury, although elevation alone does not confirm infarction because it may also occur in other clinical conditions. This consideration agrees with the troponin interpretation review, which emphasises interpreting troponin concentrations through serial measurements and their relationship with clinical manifestations (1). The BNP review also examined BNP's usefulness in patients with dyspnoea, helping distinguish possible cardiac and non-cardiac causes (2). These contributions indicate that no marker provides sufficient diagnostic information in isolation; interpretation requires integration of clinical parameters and complementary procedures. Inflammatory and metabolic mechanisms also play an important role in cardiovascular disease progression.

High-sensitivity C-reactive protein characterises systemic inflammatory activity and complements coronary risk assessment, while lipoprotein(a) provides information on atherosclerotic susceptibility associated with hereditary factors. From the perspective of inflammatory pathophysiology, the analysis of atherosclerotic inflammation explained the contribution of vascular inflammation to atherosclerotic progression (9). This interpretation also relates to the contributions of the analysis of atherosclerotic inflammation, which describes interactions among lipids, immune cells and the arterial wall during atherosclerotic progression (9). The LDL cholesterol research highlights the relationship between LDL cholesterol and atherosclerotic cardiovascular disease (12). Joint assessment of inflammatory, lipid and hereditary indicators therefore helps explain cardiovascular risk as a multifactorial phenomenon. Incorporating artificial intelligence and machine learning expands the possibilities for integrating clinical and biochemical information to estimate cardiovascular susceptibility.

A review on precision cardiovascular medicine examined integration of pharmacogenomics, biomarkers and machine learning; this approach offers a broad range of variables but requires verification of usefulness outside the training data (13). Nevertheless, these systems' computational capability does not, by itself, guarantee greater clinical accuracy. In agreement with the scientific statement on model assessment, the reliability of estimates depends on calibration, discrimination, data quality and validation in independent populations (16). Incorporating predictive algorithms therefore requires assessment criteria to determine actual usefulness and prevent automated estimates from being interpreted independently of each patient's circumstances. The relationship among biomarkers, cardiovascular prediction and therapeutic selection demonstrates the importance of individualised assessment.

Contributions from the precision cardiovascular medicine review (13) emphasise integration of biological and technological information as a foundation of precision medicine, while the lipoprotein(a) study (3) specifically examined lipoprotein(a) as an inherited and underdiagnosed risk factor. From this perspective, combining clinical indicators and predictive tools supports identification of susceptibility profiles and provides complementary criteria for guiding interventions concerning lipid control, hypertension and thrombotic event prevention. However, therapeutic selection

continues to depend on professional assessment, clinical conditions, contraindications and applicable medical recommendations. The principal contribution of this integration is to expand available information to anticipate cardiovascular abnormalities and support preventive and therapeutic decisions better adapted to individual characteristics.

CONCLUSIONS

Integrating clinical biomarkers and predictive models contributes meaningfully to early diagnosis of cardiovascular diseases through their ability to identify biological abnormalities, recognise susceptibility conditions and broaden individual risk estimation. Indicators such as cardiac troponins, high-sensitivity C-reactive protein, BNP and lipoprotein(a) provide complementary information on myocardial injury, inflammatory activity, cardiac function and atherosclerotic predisposition. Their use strengthens clinical assessment, particularly during the early stages of cardiovascular abnormalities.

Combining them with risk equations and AI-based tools supports a broader interpretation of the conditions involved in cardiovascular disease onset and progression. Predictive models integrate clinical and biochemical variables and electronic records to recognise patterns associated with possible cardiovascular events. However, usefulness depends on data quality, algorithm calibration and validation in different populations, all necessary for reliable and clinically relevant estimates.

Regarding therapeutic decisions, integration of biomarkers and predictive systems provides complementary information to establish individual risk profiles and guide preventive and clinical measures. This combination supports interventions concerning lipid concentration control, blood pressure management and prevention of cardiovascular complications. It also supports an approach linked to precision medicine, in which each patient's biological and clinical characteristics are relevant to selecting therapeutic alternatives.

Clinical biomarkers and predictive models strengthen early diagnosis of cardiovascular diseases by complementing identification of initial abnormalities with individualised risk estimation. Their application broadens the information available to guide preventive and therapeutic decisions, although it does not replace health professionals' judgement or comprehensive patient assessment. Incorporation therefore requires informed use, clinical validation and coordination with routine diagnostic procedures to support timely decisions adapted to each person's particular circumstances.

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ETHICAL CONSIDERATIONS

The study was conducted through a literature review based exclusively on previously published documentary sources. It did not involve direct participation of humans or animals, interventions, biological samples or access to identifiable personal data. Consequently, it did not require ethics committee approval or informed consent. The research process observed the principles of scientific integrity, appropriate authorship attribution, rigorous citation and responsible use of consulted sources.

DECLARATION OF ARTIFICIAL INTELLIGENCE (AI) USE

The authors state that the artificial intelligence tool was used exclusively to support article organisation and text translation. Its use did not determine scientific content, study design, source selection, data analysis, interpretation of results or conclusions. Full responsibility for the content remains with the authors.

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CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest related to this study and that all procedures followed comply with the journal's ethical standards. They also confirm that this work is original and has not been published, in whole or in part, in any other publication.

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