Revista de Ciencias Médicas de Pinar del Río

ISSN: 1561-3194 **RNPS:** 1877
Rev Ciencias Médicas. 2025; 29(S2): e7024

REVIEW ARTICLE

Cardiovascular effects of the use of nonsteroidal anti-inflammatory drugs in patients with a history of cardiovascular disease

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Received: December 27, 2025
Accepted: December 28, 2025
Published: December 30, 2025

Citar como: Barragan-Guillen VM, Gavilanes-Salazar LD, Acuña-Ango MA, Herrera-Villacres ER. Efectos cardiovasculares del uso de antiinflamatorios no esteroideos en pacientes con antecedentes de enfermedad cardiovascular. Rev Ciencias Médicas [Internet]. 2025 [citado: fecha de acceso]; 29(S2): e7024. Disponible en: http://revcmpinar.sld.cu/index.php/publicaciones/article/view/7024

ABSTRACT

Introduction: non-steroidal anti-inflammatory drugs are widely prescribed for pain and inflammation, but their cardiovascular safety raises concerns in patients with cardiac history.
Objective: to describe the association between diclofenac and ibuprofen and the incidence of cardiovascular events in individuals with pre-existing cardiovascular disease.
Methods: a systematic review of the scientific literature was conducted across multiple databases. The search was performed using an algorithm with keywords and Boolean operators, allowing the identification of relevant sources. The selected studies, after applying inclusion and exclusion criteria, were critically analyzed considering timeliness, methodological quality, and thematic relevance, and were integrated into the final synthesis of the review.
Development: the literature shows that diclofenac significantly increases mortality and cardiovascular events, with risks exceeding 90 % in some studies, while ibuprofen demonstrates moderate increases, close to 30 %. The mechanisms involve inhibition of cyclooxygenase-2, disruption of prostaglandin balance, and effects on blood pressure and fluid retention. Meta-analyses confirm a higher risk of myocardial infarction and sudden death with diclofenac, whereas ibuprofen, although safer, also raises the likelihood of acute coronary syndrome and stroke with prolonged use.
Conclusions: the comparative analysis confirms that diclofenac presents a more unfavorable cardiovascular profile than ibuprofen, especially in patients with cardiac history. It is recommended to limit its prescription and prioritize safer alternatives, always considering individual risk factors and the need for rational use of anti-inflammatory drugs.

Keywords: Anti-Inflammatory Agents, Non-Steroidal; Physiological Effects of Drugs; Cardiovascular Diseases.

ISSN: 1561-3194 **RNPS:** 1877
Rev Ciencias Médicas. 2025; 29(S2): e7024

INTRODUCTION

Nonsteroidal anti-inflammatory drugs (NSAIDs) are a class of medications used to relieve pain, reduce inflammation, and control fever. They are classified into two types based on cyclooxygenase (COX) selectivity: non-selective (COX-1) and selective (COX-2) inhibitors. These drugs act by inhibiting the arachidonic acid (AA) metabolic pathway, which is involved in the production of prostaglandins—substances responsible for inflammation and pain.[1] NSAIDs are effective in treating conditions such as arthritis, osteoarthritis, musculoskeletal disorders, and various forms of pain. However, prolonged use or high doses may increase the risk of adverse effects, including gastrointestinal, cardiovascular, and renal complications in certain patients. Among the most commonly used NSAIDs are diclofenac and ibuprofen.[2]

Diclofenac is known for its rapid analgesic action, which begins approximately 60 minutes after oral administration. It has been observed to be effective in postoperative settings, providing excellent relief from pain and inflammation. However, diclofenac alone does not fully alleviate pain or inflammation in all patients. Therefore, clinicians often prescribe it alongside pharmacological adjuvants such as paracetamol or vitamin B to enhance efficacy and avoid high doses, which could increase the risk of heart failure in patients with pre-existing cardiovascular disease.[3]

Ibuprofen is another widely used NSAID with analgesic properties similar to diclofenac. It belongs to the non-selective COX inhibitor group and is recognized for its high efficacy and relatively favorable safety profile compared to other NSAIDs. Nevertheless, when used inappropriately—particularly at high doses over prolonged periods—it has been associated with increased risk of adverse cardiovascular events, including myocardial infarction, stroke, and arterial hypertension.[4]

Given that pain and inflammation are common symptoms significantly affecting the quality of life of millions worldwide, NSAIDs such as diclofenac and ibuprofen represent a widely used therapeutic class. However, growing evidence regarding the cardiovascular risks associated with certain NSAIDs—particularly diclofenac—has sparked considerable debate about their safe and effective use in clinical practice.[5]

The mechanism by which diclofenac increases cardiovascular risk involves inhibition of COX-2, which plays a crucial role in regulating platelet aggregation and vasodilation.[6] This can lead to increased thrombosis and vasoconstriction.[7] Additional contributing factors include sodium and water retention, elevated blood pressure, and interactions with antihypertensive medications, all of which shape diclofenac's unfavorable cardiovascular risk profile.[8]

Patients with prior cardiovascular disease face heightened health risks, as cardiovascular diseases (CVD) directly affect blood vessels, the heart, and cardiac rhythm.[9] These conditions are associated with an increased risk of hypertension and heart failure, particularly in relation to NSAID use—both selective and non-selective. Inhibition of COX pathways by these drugs leads to reduced vasodilation (with COX-2 inhibitors) and promotes platelet aggregation and vasoconstriction (with traditional NSAIDs).[10]

ISSN: 1561-3194 **RNPS:** 1877
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Therefore, in patients with such histories, NSAID use should be minimized or avoided whenever possible. Instead, it is essential to seek alternative treatments with more favorable safety profiles. One such alternative is opioids—a class of medications primarily used for pain relief. These drugs act on the central nervous system by binding to specific receptors to block pain signals and induce feelings of pleasure and well-being. However, their use must be carefully monitored due to their high potential for dependence and addiction.[11] Given these considerations, this review was conducted with the objective of describing the association between diclofenac and ibuprofen and the incidence of cardiovascular events in individuals with pre-existing cardiovascular disease.

METHODOLOGY

This study was designed as a systematic literature review following the PRISMA 2020 guidelines and Cochrane systematic review standards to ensure transparency, reproducibility, and methodological rigor. The search period spanned from 2010 to 2024 to capture the most recent evidence on the cardiovascular effects of NSAIDs—specifically diclofenac and ibuprofen—in patients with a history of cardiovascular disease.

Information sources included widely recognized biomedical databases: PubMed/MEDLINE, LILACS, SciELO, ScienceDirect, Cochrane Library, and Google Scholar. Secondary references from selected articles and grey literature from institutional repositories were also reviewed to broaden identification of relevant studies and minimize publication bias.

The search strategy employed an algorithm combining keywords and Boolean operators. MeSH and DeCS terms such as "Diclofenac," "Ibuprofen," "Cardiovascular Risk," "Myocardial Infarction," "Non-steroidal Anti-inflammatory Drugs," and "COX-2 Inhibitors" were combined using AND and OR operators to maximize sensitivity and specificity. Publications in Spanish, English, and Portuguese were included to integrate evidence from diverse clinical and linguistic contexts.

Inclusion criteria encompassed original articles, clinical trials, cohort studies, meta-analyses, and systematic reviews published within the defined timeframe that directly addressed the relationship between diclofenac, ibuprofen, and cardiovascular events in patients with prior cardiac history. Duplicates, articles without full access, irrelevant documents, publications prior to 2010, as well as letters, editorials, clinical practice guidelines, theses, and books were excluded.

The selection process occurred in several phases: initial title and abstract screening to exclude non-relevant studies, followed by full-text evaluation of potentially eligible articles. Initially, approximately 75 records were identified; after removing duplicates and applying exclusion criteria, the sample was reduced to 28 articles; finally, nine studies were included in the qualitative synthesis. The procedure was documented using a PRISMA flow diagram (Figure 1), illustrating each selection stage.

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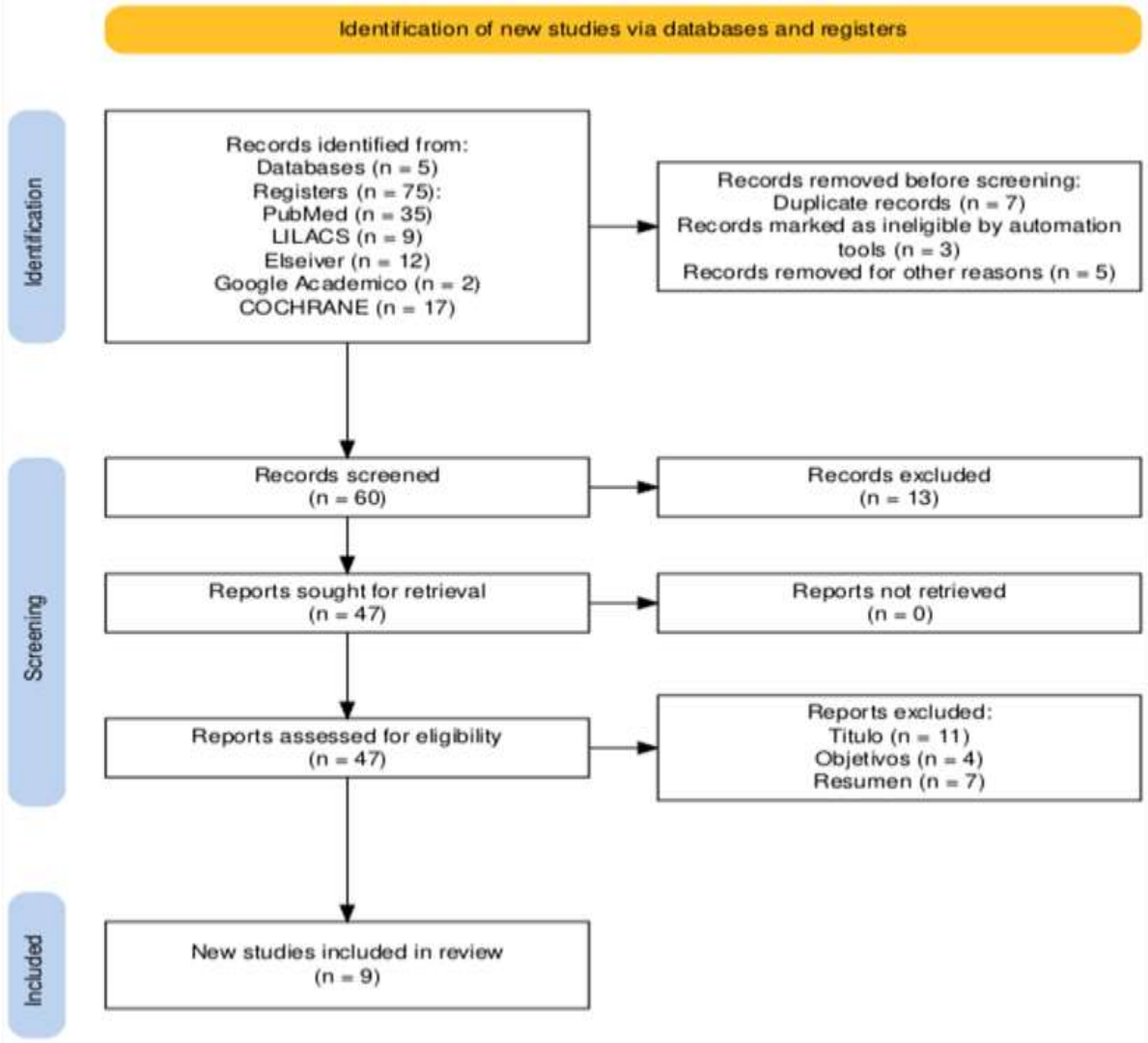

Fig. 1 Flowchart.

Data extraction and analysis were performed systematically, collecting key variables such as author, publication year, methodological design, population characteristics, type of pharmacological intervention, cardiovascular outcomes, and main findings. Information was organized into comparative matrices to facilitate interpretation. A qualitative synthesis was conducted, as methodological and outcome heterogeneity precluded formal meta-analysis. This approach enabled integration of available evidence and provided a critical, up-to-date overview of cardiovascular risks associated with diclofenac and ibuprofen use in patients with prior cardiovascular disease.

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DEVELOPMENT

Diclofenac is an NSAID whose analgesic effect begins approximately 60 minutes after oral administration. It is considered effective for managing postoperative pain and inflammation. However, diclofenac alone does not always provide complete symptom relief; thus, clinicians often combine it with adjuvant medications such as paracetamol or vitamins to enhance efficacy and avoid high doses that could increase the risk of heart failure in patients with pre-existing cardiac conditions.

Table 1. Comparison of ibuprofen and diclofenac regarding cardiovascular risks.

Source	Ibuprofen (cardiovascular effects)	Diclofenac (cardiovascular effects)	Comparison
Schmidt et al., (2017)[7]	Ibuprofen users had 40% lower risk of major cardiovascular events (heart attack, stroke). All-cause mortality was 50% lower vs. diclofenac users.	Diclofenac users had double the risk (100% higher) of major cardiovascular events vs. non-users. • Heart attack: +100% • Heart failure: +80% • Venous thromboembolism: +390% (nearly 4×)	Ibuprofen associated with lower overall risk and mortality. Diclofenac shows a markedly unfavorable cardiovascular profile with significant increases in both fatal and non-fatal events.
Schjerning et al., (2013)[12]	Ibuprofen use associated with 34% higher risk of cardiovascular death (HR 1.34, 95% CI: 1.26–1.44).	Diclofenac use associated with 96% higher risk of cardiovascular death (HR 1.96, 95% CI: 1.79–2.15).	Diclofenac carries greater cardiovascular mortality risk than ibuprofen. • Diclofenac: +96% (HR 1.96) • Ibuprofen: +34% (HR 1.34)
Schmidt et al., (2024)[13]	New ibuprofen users showed 31% increased risk of adverse cardiovascular events (HR 1.31). No significant association found for continuous use (HR 1.03).	New diclofenac users (n=1,894,834) had 50% higher risk of major adverse cardiovascular events (MACE) vs. non-users (n=3,789,617) (IRR 1.53, 95% CI: 1.43–1.63). • Myocardial infarction: +54% (IRR 1.54) • Cardiac death: +92% (IRR 1.92)	Diclofenac poses higher cardiovascular risk for new users than ibuprofen (63% vs. 31%). No significant risk increase with continuous ibuprofen use, but diclofenac showed an 11% increase.
Fosbøl et al., (2009)[14]	Risk of death/myocardial infarction with ibuprofen: RR 1.01 (95% CI: 0.96–1.07)—comparable to non-users.	Diclofenac associated with significantly higher risk of death and MI: RR 1.63 (95% CI: 1.52–1.76).	Diclofenac showed significantly higher cardiovascular risk than ibuprofen. Ibuprofen: no significant increase (RR 1.01); Diclofenac: +63% (RR 1.63).
Thöne et al., (2017)[15]	Relative risk of MI: 1.54 (95% CI: 1.43–1.65)—54% increase in current ibuprofen users vs. non-users.	Relative risk of MI: 1.43 (95% CI: 1.34–1.52)—43% increase in current diclofenac users. Risk similar in patients with/without major CV risk factors.	Ibuprofen showed higher MI prevalence than diclofenaco. • Ibuprofen OR: 1.54 (54%) • Diclofenac OR: 1.43 (43%)
Muñoz Olmo et	Moderate risk with ibuprofen.	Diclofenac is among the highest-risk NSAIDs,	Diclofenac presents higher cardiovascular risk than

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Rev Ciencias Médicas. 2025; 29(S2): e7024

al., (2018)[16]	Increased risk of acute coronary syndrome after 5 years (peak in year 2: OR 1.63). Elevated stroke risk (HR 1.23).	associated with fatal and non-fatal acute coronary syndrome.	ibuprofen, which has moderate risk.
Krötz et al., (2010)[17]	No conclusive association with significant MI risk.	Increases MI risk in both high-risk and healthy individuals. Promotes cardiovascular events and arterial clots—a risk specific to diclofenac, not seen with other NSAIDs. Alters prostaglandin balance, favoring thrombosis.	MI risk primarily linked to diclofenac, not traditional NSAIDs like ibuprofen. Diclofenac carries higher cardiovascular risk.

On the other hand, ibuprofen is a widely used NSAID for treating pain, inflammation, and fever. Its primary mechanism of action involves inhibition of prostaglandin synthesis. However, prolonged use of ibuprofen and other NSAIDs may also be associated with an increased risk of adverse cardiovascular events. Given the widespread prescription and use of diclofenac and ibuprofen, any significant cardiovascular risk could have considerable public health implications. Therefore, understanding these risks is essential to guide prescribing recommendations—especially in patients with pre-existing cardiovascular risk factors—and to develop treatment strategies that ensure maximum efficacy with minimal patient risk.

Table 2. Comparison of mechanisms of action between ibuprofen and diclofenac.

Source	NSAID	Mechanism of Action
Altman et al., (2015)[18]	Diclofenac	Inhibits both COX-1 and COX-2 enzymes, responsible for synthesizing various prostaglandins (PGE2, PGD2, PGF2, PGI2, TXA2). This dual inhibition affects both vasodilatory/antiplatelet prostaglandins (e.g., PGI2) and pro-thrombotic/vasoconstrictive ones (e.g., TXA2). The resulting imbalance favors a pro-thrombotic, vasoconstrictive state, increasing the risk of myocardial infarction and stroke.
Tiwari et al., (2018)[19]	Ibuprofen	Widely used due to its favorable safety profile and analgesic/anti-inflammatory activity via COX-1 and COX-2 inhibition. COX-1 inhibition reduces mucosal protection and increases gastric acid secretion, potentially causing erosion, ulceration, perforation, and bleeding. Selective COX-2 inhibition may reduce endothelial prostacyclin production, increasing thrombosis and vascular event risk.

Schmidt et al.,[7] suggest that ibuprofen carries a 40 % lower cardiovascular risk compared to diclofenac, a finding supported by other studies. According to Schjerning et al.,[12] diclofenac shows significantly higher cardiovascular risk: diclofenac use was associated with a 96 % increase in cardiovascular mortality (HR 1,96, 95 % CI: 1,79–2,15), whereas ibuprofen showed a 34 % increase (HR 1,34, 95 % CI: 1,26–1,44).

Schmidt et al.,[13] also found that new diclofenac users had a 50 % higher risk of major adverse cardiovascular events compared to non-users (IRR 1,53, 95 % CI: 1,43–1.63), including a 54 % increase in myocardial infarction (IRR 1.54) and a 92% increase in cardiac death (IRR 1,92). Although ibuprofen shows lower risk than diclofenac, diclofenac consistently demonstrates considerably higher risk—up to 100 % in some cases.

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Fosbøl et al.,[14] reported that diclofenac use was associated with a significantly higher risk of death and myocardial infarction (RR 1,63), while ibuprofen showed no significant increase (RR 1,01), indicating diclofenac's substantially greater cardiovascular risk.

Thöne et al.,[15] reported higher relative risk of myocardial infarction for both ibuprofen (54 %) and diclofenac (43 %), with similar risks regardless of pre-existing cardiovascular risk factors. In contrast, Muñoz Olmo et al.,[16] concluded that diclofenac is among the NSAIDs with the highest cardiovascular risk, strongly associated with fatal and non-fatal acute coronary syndrome, whereas ibuprofen presented only moderate—and comparatively lower—risk.

Krötz et al.,[17] reported that diclofenac increases cardiovascular event risk in both patients with and without prior cardiac history, showing greater risk than traditional NSAIDs like ibuprofen, which demonstrated no significant risk. Notably, arterial clot formation appears to be a characteristic unique to diclofenac among NSAIDs.

The primary factor increasing these risks lies in their mechanisms of action. According to Altman et al.,[18] diclofenac inhibits both COX-1 and COX-2, disrupting the synthesis of various prostaglandins—including vasodilatory/antiplatelet (PGI2) and pro-thrombotic/vasoconstrictive (TXA2) types. This dual inhibition can create an imbalance favoring a pro-thrombotic, vasoconstrictive state, thereby increasing the risk of myocardial infarction and stroke.

Tiwari et al.,[19] note that diclofenac's cardiovascular effects may also stem from mitochondrial dysfunction, leading to elevated reactive oxygen species (ROS) that damage cellular proteins.

CONCLUSIONS

Scientific evidence shows some discrepancies but generally indicates that diclofenac carries a higher cardiovascular risk than ibuprofen, although both nonsteroidal anti-inflammatory drugs can increase the likelihood of adverse events in patients with prior cardiac history. Diclofenac, effective for postoperative pain, is often combined with other drugs to reduce high doses that might promote heart failure, while ibuprofen—widely used for pain, inflammation, and fever—is also associated with cardiovascular risk during prolonged use. Given their widespread prescription, understanding these risks is essential to guide therapeutic decisions and ensure effective treatments with minimal danger to public health.

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