



Nonsteroidal anti-inflammatory drugs and aspirin in complicated diverticular disease

Ulises Rodríguez-Wong,* Ulises Rodríguez-Medina**

* Cirujano Gastroenterólogo y Coloproctólogo. Hospital Ángeles Health System. Maestro en Ciencias de la Salud, Doctor en Ciencias Sociales y Administrativas.
** Médico Residente de Medicina Interna. University of New Mexico Hospital, USA. Doctor en Ciencias Sociales y Administrativas.

Medicamentos antiinflamatorios no esteroides y aspirina en la enfermedad diverticular complicada

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Diverticular disease is highly prevalent in Western countries, affecting up to 60% of individuals over 60 years of age.¹ Although most patients remain asymptomatic, a substantial proportion develops complications such as acute diverticulitis and diverticular bleeding.²

Nonsteroidal anti-inflammatory drugs (NSAIDs) and aspirin are widely prescribed for analgesic, anti-inflammatory, and cardioprotective purposes. However, cumulative evidence suggests that these medications may increase the risk of diverticular complications.³

Diverticulosis predominantly affects the sigmoid and descending colon and is associated with aging; low-fiber diets, obesity, and physical inactivity.⁴ Approximately 10-25% of patients with diverticulosis develop diverticulitis during their lifetime.⁵

Diverticular bleeding is the most common cause for lower gastrointestinal hemorrhage in adults, accounting for up to 40% of cases.⁶ Risk factors include hypertension, chronic kidney disease, anticoagulant therapy, and NSAID use.⁷

Aspirin and NSAIDs exert their effects through inhibition of cyclooxygenase (COX) enzymes, leading to decreased prostaglandin synthesis.⁸ While this mechanism mediates analgesic and anti-inflammatory effects, it also compromises gastrointestinal mucosal defense.

Aspirin irreversibly inhibits COX-1 and COX-2, whereas most NSAIDs produce reversible inhibition.⁹ Both drug classes reduce mucosal blood flow, impair epithelial regeneration, and increase intestinal permeability.¹⁰

Several mechanisms have been proposed to explain the association between NSAID use and diverticular disease complications. Mucosal injury: NSAIDs increase intestinal permeability and promote epithelial damage.¹¹ Prostaglandin suppression: Reduced prostaglandin levels impair mucosal protection and healing.¹² Microvascular damage: Platelet inhibition and endothelial injury may promote bleeding.¹³ Altered microbiota: NSAIDs may induce dysbiosis, contributing to inflammation.¹⁴ These mechanisms facilitate inflammation, ulceration, perforation, and hemorrhage in the diverticula.

Several epidemiological studies have demonstrated a strong association between NSAID use and diverticulitis. In a large prospective cohort study, *Strate, et al.*, reported that regular NSAID use increased the risk of developing diverticulitis by 72%.¹⁵

Similarly, *Aldoori, et al.*, observed a higher incidence of complicated diverticulitis, including perforation and abscess formation, among chronic NSAID users.¹⁶ A meta-analysis by *Morris, et al.*, confirmed that NSAID exposure significantly increased the risk of diverticular perforation.¹⁷

Correspondencia:

Dr. Ulises Rodríguez-Wong
Tepic Núm. 113, Int. 611. Col. Roma Sur. C.P. 06760. Alcaldía Cuauhtémoc, CDMX, México
Correo electrónico: ulisesromed@prodigy.net.mx

Aspirin use is particularly associated with diverticular hemorrhage. A prospective study involving more than 47,000 men demonstrated that regular aspirin use was associated with a twofold increased risk of diverticular bleeding.¹⁵

Yuhara, *et al.*, in a systematic review, reported that both, low-dose and high-dose aspirin increased bleeding risk, with a dose-dependent effect.¹⁸ The antiplatelet properties of aspirin, combined with mucosal injury, are believed to be central to this association.¹⁹

The relationship between NSAIDs, aspirin, and diverticular complications has important clinical implications. Patients with known diverticulosis, advanced age, prior diverticulitis, or bleeding history represent high-risk populations.²⁰

Alternative analgesics such as acetaminophen may be preferred in susceptible individuals.²¹ For patients requiring antiplatelet therapy, risk stratification and gastroprotective strategies should be considered.²²

CONCLUSIONS

Current evidence strongly supports an association between aspirin or NSAID use and increased risk of diverticulitis and diverticular bleeding. These drugs compromise intestinal mucosal integrity and promote inflammation and hemorrhage. Careful patient selection, dose optimization, and preventive strategies are essential to minimize adverse outcomes.

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