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Mini-Review Article

Beyond Pain Relief: Exploring the Hidden Renal Consequences of NSAID Therapy

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ABSTRACT

Background: Older adults are especially vulnerable to peptic ulcers, gastrointestinal irritation, and fluid retention caused by non-steroidal anti-inflammatory drugs (NSAIDs). It is common to inappropriately prescribe NSAIDs for osteoarthritis before adequately addressing physical and functional interventions along with oral paracetamol. If an NSAID is necessary as a supplementary treatment, it should be administered at the lowest effective dosage. Ibuprofen is likely the preferred NSAID for reducing gastrointestinal side effects. A proton pump inhibitor should be considered to prevent upper gastrointestinal complications in individuals at higher risk.

Methods: NSAIDs can also lead to sodium and water retention, increased blood pressure, hyperkalemia, and swelling. Frequent or severe instances of kidney damage can lead to a gradual decline in kidney function, particularly in at-risk patients. Identifying risk factors and avoiding unnecessary long-term use of NSAIDs is essential.

Results: Additionally, it helps with dysmenorrhea. It is a reversible cyclooxygenase inhibitor sold over the counter in the UK and many other countries. At normal dosages, it causes less gastric irritation than aspirin and other NSAIDs. Children can use a suspension.

Conclusion: NSAID-related side effects include reversible renal impairment in elderly patients or those with cirrhosis, nephrotic syndrome, or heart failure. By preventing formation, it lessens the effectiveness of diuretics and antihypertensive drugs.

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Introduction

By blocking cyclo-oxygenase (COX), non-steroidal anti-inflammatory drugs (NSAIDs) prevent prostaglandin biosynthesis.

The majority of their therapeutic and undesirable effects stem from this. The production of prostaglandins and thromboxanes, which are crucial mediators of inflammation-related erythema, edema, pain, and fever, depends on COX. The enzyme exists in two primary isoforms: the constitutive form (COX-1), which is found in platelets, the stomach, the kidneys, and other tissues, and the inducible form (COX-2), which is expressed in inflammatory tissues due to cytokine stimulation and is also present in healthy organs, such as the kidneys, to a lesser degree. [1, 3].

Methods

The purpose of this work was to examine the relationship between exposure to NSAIDs and renal dysfunction. Renal hemodynamic effects, acute kidney injury, chronic kidney disease, and clinical risk factors for NSAID-associated nephrotoxicity were the four main domains examined in the literature. Relevant experimental and clinical data on selective COX-2 inhibitors and traditional non-selective NSAIDs were considered.

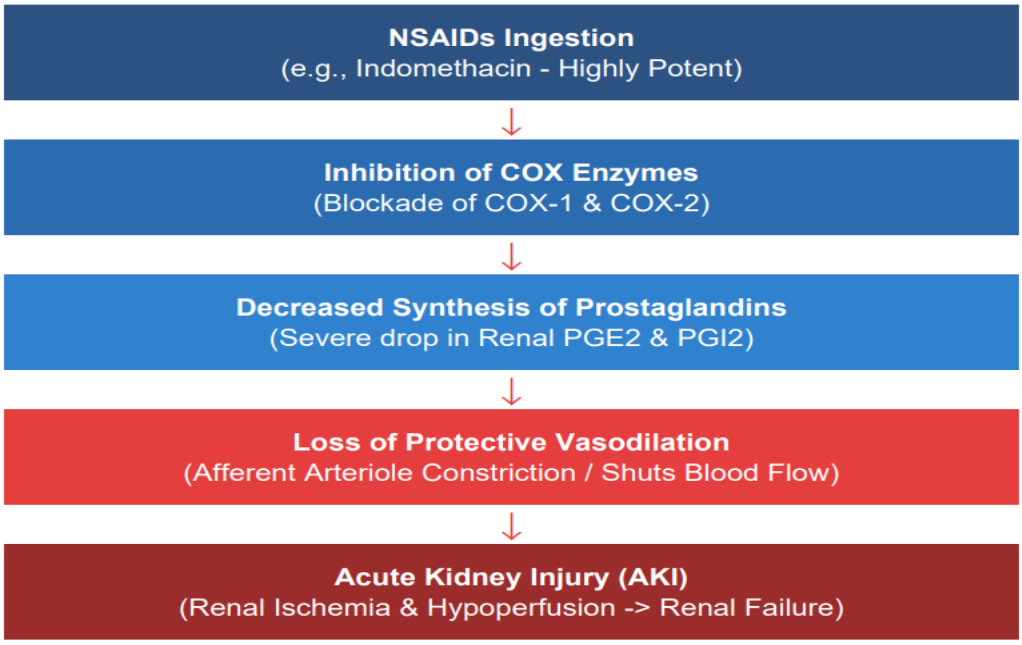
Studies detailing alterations in renal blood flow, glomerular filtration, serum creatinine, estimated glomerular filtration rate, electrolyte balance, and clinical signs of renal injury were the main focus of the review. Populations at higher risk of nephrotoxicity, such as older adults and those with pre-existing kidney disease, volume depletion, cardiovascular disease, or concurrent use of drugs that impact renal perfusion, received special attention. The biological mechanisms and clinical ramifications of NSAID-induced renal dysfunction were explained by a qualitative synthesis of the available data [7]

Results

The reviewed data show that exposure to NSAIDs can negatively impact renal function in a number of ways. Inhibition of renal prostaglandin synthesis is the most significant mechanism. Renal vasoconstriction and decreased renal blood flow result from inhibition of COX activity, which also reduces prostaglandin-mediated afferent arteriolar dilatation. In healthy individuals, the decrease in glomerular filtration might not be noticeable, but when renal perfusion is already impaired, it can become significant [15].

Renal effects of NSAIDs

- NSAIDs stop the production of prostaglandins PGE2 and PGI2, which are in charge of preserving renal blood flow.



International Journal of Medical Toxicology & Forensic Medicine

Figures 1. Pathway of NSAIDs to induce kidney injury.

- Reduced prostaglandin synthesis may lead to edema and sodium and water retention.

NSAIDs' effects on the heart

The cardiovascular protective effect of agents like aspirin, which have a very high degree of COX-1 selectivity at low doses, is believed to be caused by a decrease in TXA2 production.

NSAIDs' effects on the bronchi

NSAIDs prevent the synthesis of prostaglandins but not leukotrienes because they are cyclooxygenase inhibitors. Because prostaglandin synthesis inhibition can lead to a shift toward leukotriene production and raise the risk of asthma exacerbations, NSAIDs should be used cautiously in asthmatic patients [9].

NSAIDs and pregnancy

If analgesic effects are required during pregnancy, acetaminophen is the recommended medication.

NSAIDs should normally be avoided during the third trimester due to the possibility of the ductus arteriosus closing too soon.

Discussion

NSAIDs' mode of action • Aspirin and other NSAIDs have three main therapeutic effects: they reduce fever (antipyretic effect), pain (analgesic effect), and inflammation (anti-inflammatory effect). For each of these purposes, not all NSAIDs are equally effective.

• NSAIDs' anti-inflammatory properties

• Cyclooxygenase inhibition reduces prostaglandin synthesis, which modifies prostaglandin-mediated aspects of inflammation.

• Gastric cytoprotection, vascular homeostasis, platelet aggregation, and reproductive and renal functions are all regulated by the constitutive enzyme cyclooxygenase-1 (COX-1).

• Tissues like the brain, kidney, and bone constitutively express the inducible enzyme cyclooxygenase-2 (COX-2). Chronic inflammatory states increase its expression at other sites. Inhibition of COX-2 is thought to underlie the anti-inflammatory and analgesic effects of NSAIDs, whereas inhibition of COX-1 is responsible for preventing cardiovascular events and most adverse events.

Inhibition of COX-2 is thought to lead to the anti-inflammatory and analgesic actions of NSAIDs [15].

Conclusion

- To reduce gastrointestinal distress, NSAIDs should be taken with food or liquids.
- To avoid NSAID-induced ulcers, proton pump inhibitors or misoprostol should be used concurrently with NSAIDs in patients who are at high risk for GI events.
- Patients with established cardiovascular disease should not use NSAIDs other than aspirin.

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Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this manuscript. All authors contributed to the manuscript and approved the final version.

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