to these problem lists. They also need to routinely review pending tests and treatments, including those ordered during the on-call period, as part of their daily workflow. One practical and systematic way of doing this is to review the physician orders section of the patient medical record on a daily basis and to decide whether tests and treatments are still necessary. On a systems level, possible solutions include modifying electronic medical records systems to include reminders of pending diagnostic tests, or hiring patient navigators to keep better track of pending tests. Most important, health care teams should engage patients and families to participate actively in the coproduction of their care plans and empower them to speak up in situations in which tests are no longer necessary.

In summary, our case serves as a reminder to think carefully about ordering appropriate diagnostic tests and the importance of cancelling unnecessary tests when they are no longer indicated.


Conflict of Interest Disclosures: None reported.

LESS IS MORE

Nonsteroidal Anti-inflammatory Drug Use in a Patient With Hypertension
A Teachable Moment

Story From the Front Lines
A man in his 60s with hypertension, hyperlipidemia, and knee osteoarthritis presented with fatigue, decreased exercise tolerance, and worsening bilateral leg swelling. For 5 years, he had been prescribed lisinopril, simvastatin, and meloxicam as needed for pain. Over the past 3 months, he had been taking meloxicam, 15 mg daily, for worsening knee pain following a minor fall that led to a persistent sense of discomfort that limited him from pursuing recreational activities like biking and running but did not otherwise prevent him from performing his activities of daily living. He had not tried acetaminophen or other pain relievers before. During this same time, his lisinopril dose was doubled to 20 mg daily because his blood pressure had been slowly rising.

Basic chemistry panel and urinalysis were ordered, revealing a creatinine level of 3.51 mg/dL and blood urea nitrogen level of 61 mg/dL; 6 months previously, these levels had been 0.96 mg/dL and 18 mg/dL, respectively (to convert creatinine to micromoles per liter, multiply by 88.4). Both meloxicam and lisinopril were withheld, and the patient’s blood pressure was closely monitored. After 3 months, his swelling and sense of fatigue had decreased, but his creatinine level remained elevated at 2.17 mg/dL. At that time, he was started on amlodipine, 5 mg daily, and referred to the nephrology service for treatment of probable nonsteroidal anti-inflammatory drug (NSAID)-induced nephropathy. There, our patient asked “Doc, why did this happen to me? Is there something I could have done to save my kidneys?”

Teachable Moment
Nonsteroidal anti-inflammatory drugs are some of the most commonly used medications in the United States today, with over 19% of the noninstitutionalized civilian population using at least 1 on a regular basis. Their analgesic and anti-inflammatory properties are well known, making them ubiquitous in the treatment of chronic pain, arthritis, and other musculoskeletal conditions. However, they are not without their drawbacks. Their propensity to cause peptic ulcers, worsen cardiovascular and cerebrovascular outcomes, and impair renal function, especially when administered with many antihypertensive agents, has more recently come to light in major epidemiological studies.2,3 Therefore, it is important to consider both the risks and benefits of NSAID use when individualizing pain management strategies.

Hypertension is a leading cardiovascular risk factor worldwide, with an incidence that increases sharply with advancing age. In the United States, over 70% of those older than 70 years have been diagnosed as having hypertension, and most are treated with pharmaceutical agents.4 While most cases are deemed “essential” without a single, clearly identifiable physiologic cause, a meta-analysis4 has demonstrated that NSAID use increases blood pressure by an
average of 5 mm Hg, and, in those with established hypertension, by as much as 14 mm Hg.

The mechanism by which NSAIDs promote hypertension is not fully understood but is believed to be due to the inhibition of prostaglandin synthesis, which not only blocks mediators of pain and inflammation but also prevents natriuresis and local renal vasodilation, mechanisms that help to regulate blood pressure. In addition, NSAIDs elevate levels of serum aldosterone, which also contributes to sodium retention and, thus, to edema and hypertension. It is important to note that both nonselective and COX-2-selective inhibitors contribute to these effects, although the magnitude depends on the individual drug in question.\(^2\) As in this case, while it is impossible to know with certainty the etiology of renal failure, combining NSAIDs and angiotensin-converting enzyme (ACE) inhibitors in older patients can be risky.

When faced with poorly controlled blood pressure, the natural tendency is to simply augment antihypertensive therapy, but, without addressing the underlying issue, this may not be a viable option. Because NSAIDs increase aldosterone levels, they render ACE inhibitors and aldosterone receptor blockers (ARBs) less effective. Furthermore, the addition of antihypertensive agents, including ACE inhibitors, ARBs, diuretics, and β-blockers, can further hinder renoprotective responses, especially in the elderly who already have reduced physiologic reserve.\(^2\) In particular, the combination of NSAIDs, ACE inhibitors, and diuretics has been shown to increase the risk of acute kidney injury by 31%.\(^5\)

As this case illustrates, it is important to individualize pain management strategies. Alternatives, especially nonpharmacologic approaches, such as physiotherapy and cognitive behavioral therapy, should be emphasized. Capsaicin and menthol have also been proven to be effective in the management of arthritic pain, as well as topical diclofenac, which seems to be associated with fewer serious adverse effects compared with systemic NSAIDs, although long-term effects have not yet been studied.\(^4\) And other analgesics, such as acetaminophen, tramadol, or opiates, which carry their own risks, may be considered in the appropriate clinical context. When NSAID use is unavoidable in higher risk patients, such as those with hypertension, heart disease, or chronic kidney disease, we should minimize risks by limiting cumulative dosage, avoiding polypharmacy, and maintaining close clinical monitoring.\(^4\)

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