Slow-release NSAIDs pose greater risk of GI bleeding

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A study conducted at the Spanish Centre for Pharmacoepidemiological Research revealed that the risk of gastrointestinal complications due to nonsteroidal anti-inflammatory drug (NSAID) use varies by specific NSAID administered and by dosage. The study further determined that NSAIDs with a long half-life or slow-release formulation are associated with a greater risk of GI bleeding or perforation. Study findings are published in the June issue of *Arthritis & Rheumatism*, a journal of the American College of Rheumatology.

NSAIDs such as Advil, Motrin and Aleve, are drugs that treat pain and inflammation by blocking the action of two cyclooxygenase (COX) enzymes. COX-2 promotes inflammation, but COX-1 protects the lining of the stomach. If an NSAID inhibits both COX-1 and COX-2, GI bleeding and ulcers can result.

According to the American College of Gastroenterology, it has long been recognized that persons using NSAIDs are at a significantly increased risk of gastrointestinal complications, for instance, injury to the intestinal lining that can result in ulcers and/or gastrointestinal bleeding. With millions taking NSAID pain medications every day, it is estimated that more than 100,000 Americans are hospitalized each year and between 15,000 and 20,000 Americans die each year from ulcers and gastrointestinal bleeding linked to NSAID use.

To reduce the morbidity associated with NSAIDs, specific estimates for individual drugs and individual groups of patients with different risk

profiles are needed. This study assessed the risk of upper GI bleeding and perforation among individual NSAIDs and analyzed the correlation between this risk and the degree of inhibition of whole blood COX-1 and COX-2 in vitro.

The research team conducted a systematic review of nine observational studies on NSAIDs and upper GI bleeding/perforation published between 2000 and 2008. The article criteria was 1) report case-control or cohort studies evaluating traditional NSAID or coxib use and upper GI bleeding/perforation in the general population, and 2) provide either an estimate or enough data to estimate a relative risk comparing NSAID users with nonusers. The pooled relative risk (RR) estimates of upper GI bleeding/perforation for individual NSAIDs was calculated, as well as whether the degree of inhibition of whole blood COX-1 and COX-2 in vitro by average circulating concentrations predicted the RR of upper GI bleeding/perforation.

The analysis suggests that NSAID-associated upper GI toxicity is the result of two pharmacologic features: drug exposure and sparing of COX-1 activity. These findings support the notion that there are multifactorial determinants in the risk of upper GI bleeding/perforation among NSAID users, including clinical background, use of concomitant medications, or a possible genetic susceptibility.

Study leader Luis A. García Rodríguez, M.D. states, "We showed that persistent exposure to the drug is an important independent determinant; in fact, drugs with a long half-life or slow-release formulation were associated overall with a greater risk than NSAIDs with a short half-life. We observed the lowest GI toxicity with coxibs, i.e., celecoxib and rofecoxib, which supports the notion that sparing of COX-1 in the GI tract and possibly in platelets translates clinically to a lower upper GI risk."

Medical Xpress

More information: "Variability Among Nonsteroidal Antiinflammatory Drugs in Risk of Upper Gastrointestinal Bleeding." Elvira L. Massó González, Paola Patrignani, Stefania Tacconelli, and Luis A. García Rodríguez, Arthritis & Rheumatism; Published Online: February 22, 2010 (DOI: 10.1002/art.27412); Print Issue Date: June 2010.

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