Low-Dose of Aspirin, Gastroprotection and *Helicobacter Pylori* Erradication

To the Editor:

The editorial by Lanas y Ferrández’ on the use of gastric protection in patients on low-dose aspirin therapy that recently appeared in the REVISTA is very interesting. The significant morbidity from cardiovascular disease in Western societies, the fact that aspirin has been shown to be effective in the prevention of coronary or cerebrovascular events, together with the authors’ experience on this subject makes the article of great interest. Nevertheless, we would like to make some comments on the contents of this editorial. We believe that until more studies are done, we must take into account other risk factors for the develop of complications in patients on aspirin therapy who have a history of ulcerative disease or digestive hemorrhage, who are taking non-steroidal anti-inflammatory medication (NSAIDs) in conjunction with aspirin, and who are infected with *Helicobacter pylori*. Age of 60 years or greater and corticoid or anticoagulant therapy are risk factors that must be taken into account when adding a protective agent for the gastric mucosa, as is advised in patients treated with classic NSAIDs.

On the other hand, although we agree with the recommendation to use proton pump inhibitors as preventative measure for patients on chronic aspirin therapy, we do not think it accurate to conclude that the eradication of *H. pylori* is recommended in these patients. Studies on the subjects on the use of NSAIDs at the usual doses are controversial, and it has even been suggested that *H. pylori* may have a protective effect against lesions caused by NSAIDs. In the recommendations of the Conferencia Española de Consenso3 the inadvisability of eradicating the bacteria in asymptomatic patients taking NSAIDs was mentioned, and it was also recommended that in the presence of a concomitant history of ulcer, the bacteria should be protected with a proton pump inhibitor, and to wait until NSAIDs are no longer being taken before eradicating the bacteria. As noted in the editorial, there are few current studies on the use of low-dose aspirin, with few patients, short followup periods; and some of which are only abstracts and report conflicting results. Many questions are raised by accepting the recommendation to eliminate *H. pylori* in these patients: would we have to know if a patient is infected before prescribing low-dose aspirin? Should we only check if the patient has a history of ulcers? What diagnostic tests would have to be performed a breath test, serology testing for the bacteria, or endoscopy, biopsy, and urine tests? Should the eradication of the bacteria be confirmed later? Therefore, we think that it is premature and excessive to advise this course given current contraindications and without it being established which patients are involved, and when, and how it should be carried out.

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REFERENCES


Response

To the Editor:

We appreciate the interest of Drs. Zambrana, Rodriguez-González, and Puente in our editorial published in the REVISTA. Making practical recommendations is difficult when available scientific evidence is not abundant or even existent, and does not even answer specific clinician questions. In spite of this, we believe that the available studies support the ideas and concepts contained in the editorial.

We would like to point out that although acetylsalicylic acid is a NSAID, its therapeutic use is limited to low doses for preventing occlusive vascular disease. Data obtained when using NSAIDs in anti-inflammatory or analgesic doses are not entirely applicable to low-dose aspirin. The data collected on the subject confirm and stress varying degrees of risk.
At the moment, therefore, there are no studies that show age greater than 60 years or the use of corticosteroids increases the risk of hemorrhage in patients who take low-dose aspirin. On the other hand, there are data that indicate that a history of ulcer or the concomitant use of NSAIDS significantly increases the risk of hemorrhage in these patients.2-5 We must therefore limit our recommendations to those which have a scientific basis, despite the fact that it may be reasonable to assume that higher age implies greater risk. To put the limit at 60 years seems appropriate in light of the currently available information. Similarly, data on the risk with concomitant use of anticoagulants or the presence of concomitant serious illnesses is scarce, or even nonexistent. Clinical logic would dictate that although the risk is low, developing a hemorrhage can be fatal, and therefore it seems reasonable, even imperative, to add a gastroprotective agent.

The most controversial subject seems to be that of the role of Helicobacter pylori infection. The literature, to be sure (including the recommendations of panels of experts which one of the authors of this article served on)6 is confusing. The new data, however, indicates that H. pylori is a risk factor for all patients who take NSAIDs.7 There appears to be a greater consensus when reviewing the information on low-dose aspirin; currently, few researchers doubt that H. pylori is a risk factor for patients taking that medication at that dose.2-5 This means that infection must be eradicated or tested for in all patients who take low-dose aspirin. Enacting gastroprotective measures in a patient who takes NSAIDS or low-dose aspirin requires evaluation of the risk factors present. If they do exist, especially in those who have an increased risk of hemorrhage (history of ulcer or hemorrhage, concomitantly taking NSAIDs), administering a proton pump inhibitor would likely be sufficient to significantly reduce the risk of hemorrhage. Nevertheless, a history of ulcer or previous complications favors eradication of H. pylori because, in most cases, 1 week of antibiotic treatment reduces the risk of upper digestive system hemorrhage even more (especially in the case of duodenal ulcer). It is very possible that future studies will indicate, from the risk/benefit perspective, that subpopulations require eradication of H. pylori as the only therapeutic measure. At this time, however, the most reasonable practical suggestion is to prescribe a proton pump inhibitor in the presence of risk factors and also to eradicate H. pylori if there is evidence of previous gastroduodenal ulcer. At present, this is the usual practice in gastroenterology practices in our country. The best and least difficult test to confirm the presence or absence of infection is a breath test, but other tests, including fecal testing, are also perfectly valid.

We understand that dealing with an infection like H. pylori for medical professionals not familiar with the problem results in inconsistencies in patient management, but gastroenterologists have learned the enormous benefits reported from its elimination in patients with ulcerous diathesis.

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REFERENCES