Nonsteroidal anti-inflammatory drugs (NSAIDs) are widely prescribed medicines. This fact reflects the high prevalence of rheumatic diseases. Approximately 100 million prescriptions for NSAIDs are written each year in the United States [1], let alone the large amount of preparations containing aspirin, acetaminophen or ibuprofen sold as over-the-counter drugs. Although these compounds represent a very effective class of drugs, their use is associated with a broad spectrum of untoward reactions in the liver, kidney, skin and gut.

Gastrointestinal (GI) side effects are, however, the most common adverse event encountered with this class of drugs. The gastro-duodenal damage associated with the use of NSAIDs, in particular, is an enormous clinical problem, and it has been described as ‘an emerging epidemic’ [2]. GI problems constitute a wide range of different clinical pictures, spanning from mild symptoms such as dyspepsia, heartburn and abdominal discomfort, to more serious events, like peptic ulcer and its life-threatening complications bleeding and perforation.

About 20% of patients taking NSAIDs on a daily basis has gastric erosions or ulcers when examined endoscopically and approximately 60% of patients presenting with complicated peptic ulcer disease (i.e. severe hemorrhage or perforation) are NSAID users. NSAID-induced gastropathy accounts for some 3,000 deaths per year in the US among rheumatoid arthritis patients alone [3].

Taking all the above considerations into account, I felt it worthwhile to attempt a critical overview of the available prophylactic and therapeutic options. To this end, I have asked leading experts in the field, known for their direct personal experience and scientific expertise, to compile a series of reviews to synthesize the mass of general and specific information existing in the field. Most of them participated at a Round Table that I organized during the 2nd Congress of the Mediterranean Society of Clinical Pharmacology, which took place in Venice a few years ago, and there they already did a great job. I am indebted to all of them for accepting to share with us their knowledge and for providing me with excellent manuscripts, despite the many daily commitments.

After the epidemiological background of Shorrock and Langman, the diverse and intriguing mechanisms by which NSAIDs damage gastroduodenal mucosa are discussed. Then the puzzling and still unsolved question of whether an NSAID completely devoid of GI side effects really exists is approached. Being unfortunately the answer a negative one, the two main specific options, which appear reasonable in clinical practice, are to employ mucosal protective compounds (like, for instance, PGs, the use of which is quite logical) to prevent the
occurrence of acute lesions, ulcers, hemorrhage or melena and to employ peptic ulcer healing drugs (like, for instance, antisecretory compounds), thereby allowing those arthritic patients who have had ulcers to continue taking NSAIDs. These preventive and therapeutic measures are analyzed in three different papers, one of which is devoted to the meta-analysis of the available clinical trials. Finally, the management of the often lethal NSAID-induced peptic ulcer complications (i.e. hemorrhage and perforation) is discussed. It is my hope that the present issue, which contains much of the information scattered in basic and clinical (i.e. rheumatology and gastroenterology) journals, will aid any practitioner to better understand how to accomplish the prevention or the treatment of NSAID-induced gastroduodenal damage in the individual patient.

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References


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