Gastropathy due to non-steroidal anti inflammatory drugs: pathophysiology and management

Elfizon Amir, author

Abstrak

Gastropathy refers to the damage of the epithelial cells of the gastric mucosa and disturbance of epithelial cell regeneration unaccompanied by inflammation. Gastropathy occurs due to irritation by chemical agents (such as non-steroidal anti inflammatory drugs - NSAIDs and alcohol), bile reflux, hypovolemic conditions, and chronic obstruction.

NSAIDs in general are chemical agents that cause irritation of the upper gastrointestinal tract through direct and indirect topical effects and by inhibiting prostaglandin synthesis through inhibition of COX-1 and COX-2. There are many data that demonstrates that the anti-inflammatory function of NSAIDs is mainly through inhibition of COX-2, while many of their side effects are due to inhibition of COX-1.
In general, there is a correlation between the influence of NSAID and the administered dose. The higher the dose, the higher the risk for upper gastrointestinal tract disorder. NSAID users who frequently switch drugs have a risk twice higher than those only receiving one kind of NSAID. Those who use NSAID with corticosteroids have 15 times the risk. Use ofNSAID simultaneously with anticoagulants increases the risk of bleeding from ulcer 13 times compared to control subjects. NSAID use in a patient with history of bleeding from the gastrointestinal tract is 17.2 times non-users. Smoking also increases the percentage of gastroduodenal ulcer due to NSAID.

Clinical symptoms ofNSAID gastropathy are often only dyspepsia syndrome. There is no correlation between symptoms and endoscopic findings.
The first step in the therapy ofNSAID gastropathy is termination ofNSAID administration. To treat and prevent risks of gastropathy due to NSAID, mucosal protection agents may be used. Out of the various kinds of medicine available, proton-pump inhibitors turn out to be more effective compared to H2 receptor antagonists or cytoprotectiveagents.