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MECHANISMS OF GASTROINTESTINAL TOXICITY IN ANADIN EXTRA USERS: THE ROLE OF ASPIRIN-INDUCED MUCOSAL INJURY AND PLATELET DYSFUNCTION

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Relevance. Anadin Extra is an over-the-counter analgesic that contains aspirin, paracetamol, and caffeine. Although this is effective for pain relief, there are marked side effects associated with aspirin use that can lead to erosion, ulcers, and life-threatening bleeding. The purpose of this study is to evaluate whether salicylate levels, liver enzyme activities, and platelet aggregation can be used to objectively identify patients who are at risk of developing side effects.

Aim: to evaluate the association between salicylate concentrations, platelet aggregation status, and liver function profiles, and the occurrence of GI injury in the regular Anadin Extra users, and to develop a risk stratification model.

Materials and methods. For this study, data was retrospectively collected from the clinical records of 25 regular users of Anadin Extra, where regular use is defined as patients who used ≥3 tablets/week for ≥4 weeks. The study was undertaken in one hospital, and the data collected was: serum salicylate levels (using HPLC), liver function tests (ALT and AST), platelet aggregation (arachidonic acid-induced light ssion aggregometry), fecal occult blood tests, GI symptoms, and upper GI endoscopy.

Results and their discussion. Aspirin, one of the major active ingredients in Anadin Extra, blocks the action of the enzyme cyclooxygenase. This enzyme plays a major role in the formation of prostaglandins, which are essential for maintaining the integrity of the GI tract. If the level of prostaglandins is reduced due to the action of aspirin on cyclooxygenase, the GI tract is prone to erosions, ulcers, and bleeding. This risk is further compounded in the case of Anadin Extra. In addition, aspirin also has an adverse effect on platelet function by inhibiting thromboxane A2, thereby increasing the risk of GI toxicity. In the context of the GI toxicity of Anadin Extra, it is essential to understand the above-mentioned mechanisms in order to prevent the adverse effects of the drug. GI Outcomes, among patients in the FOBT group, 22% were FOBT-positive; endoscopic examination revealed gastric erosions in 41%, gastric ulcers in 27%, and duodenal ulcers in 18%. The three-parameter model showed outstanding prediction capability. Serum Salicylate - quantifies aspirin exposure; >15 mg/dL is associated with increased risk of bleeding. Platelet Aggregation - has highest individual prediction capability. Complete platelet inhibition by aspirin (<20%) converts minor mucosal lesions into actively bleeding ulcers, validating the "double hit" hypothesis. Elevated ALT/AST helps identify patients with greater medication exposure and multi-organ involvement. The three-parameter model is useful for stratifying risk; >60% risk of bleeding in the highest risk group.

Conclusions. In this study, a clear and direct link is established between Anadin Extra and gastric ulcers. This is dose-response related and depends on the salicylate levels in the blood. The anti-platelet effect of aspirin, as demonstrated by the platelet aggregation test, is a key feature in determining the severity of the ulcer. If platelet aggregation is completely inhibited (less than 20%), bleeding occurs, and if it is not, the patient may remain symptom-free. By using levels of salicylates in the blood, platelet aggregation, and liver enzyme levels all information that can be obtained from patient case records it is possible to minimize the risk of serious gastrointestinal side effects.