Can EP4 agonist alleviate gastric lesions?

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Over 300 million patients use non-steroidal anti-inflammatory drugs (NSAIDs) in the world to treat pain, arthritis, fever and other diseases. Nearly 30% of the users suffer from gastric lesions and bleeding. To mitigate NSAIDs' adverse effects on the stomach, misoprostol, a non-selective prostaglandin E1 (PGE1) analogue, has been prescribed as the first choice for prevention of NSAID-induced injuries, but often induces severe adverse effects. There remain unmet medical needs for drugs with improved therapeutic profiles.

A research article published on November 7, 2009 in the World Journal of Gastroenterology addresses this question. A research team from United States investigated therapeutic potentials of a highly-selective EP4 agonist for treatment of a mouse gastric ulcer model in the presence or absence of indomethacin at various levels.

They found the EP4-selective agonist reduced high dose indomethacin-induced acute hemorrhagic damage and promoted mucous epithelial regeneration. Low-dose indomethacin aggravated ulcer bleeding and inflammation, and delayed the healing of the established chronic gastric ulcer. The EP4 agonist, when applied locally, not only offset indomethacin-induced gastric bleeding and inflammation, but also accelerated ulcer healing. In the absence of indomethacin, the EP4 agonist even accelerated chronic gastric ulcer healing and suppressed inflammatory cell infiltration in the granulation tissue. In vitro, the EP4 agonist protected human gastric mucous cells from indomethacin-induced apoptosis.

Their results suggest that EP4 agonists may mimic the gastric protective effects of PGE2 in the presence or absence of NSAIDs, and may show advantages over non-selective analogs such as misoprostol by minimizing adverse effects arising from activating all 4 subtype receptors of PGE2.

More information: Jiang GL, Im WB, Donde Y, Wheeler LA. EP4 agonist alleviates indomethacin-induced gastric lesions and promotes chronic gastric ulcer healing. World J Gastroenterol 2009; 15(41): 5149-5156, www.wjgnet.com/1007-9327/15/5149.asp

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