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PAPERS

Gastric mucosal contraction and vascular injury induced by indomethacin precede neutrophil infiltration in the rat

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BACKGROUND: In contrast with earlier reports that neutrophils play a primary part in non-steroidal anti-inflammatory drug (NSAID) injury to the stomach, recent evidence suggests only a secondary role for these cells. AIM: To examine whether early microscopic changes induced by indomethacin in the gastric corpus of fasted rats and the antrum of fasted-refed rats involve neutrophil infiltration. METHODS: Oral indomethacin 30 mg/kg or vehicle was given to six groups of fasted rats that were killed five, 15, and 30 minutes after dosing. Subcutaneous indomethacin 30 mg/kg was also given to six groups of fasted-refed rats that were killed one, two, and four hours later. Haematoxylin and eosin and reticulin stained sections were examined to identify mucosal architectural changes. The gastric mucosa was also examined immunohistochemically for actin, fibrin, and neutrophils. RESULTS: In both the corpus and antrum, indomethacin caused an early phase of mucosal injury that occurred prior to mucosal neutrophil infiltration. Within the superficial corpus mucosa, this phase preceded coagulative necrosis and included surface epithelial expulsion, mucosal contraction with capillary aggregation and distortion, intravascular vascular fibrin deposition, and capillary congestion. The antrum showed similar early changes except that full thickness mucosal coagulative necrosis was a predominant early finding. CONCLUSIONS: In two experimental models of NSAID gastric ulceration the mucosa undergoes early contraction, vascular fibrin deposition, and necrosis prior to neutrophil infiltration. These findings support a primary, neutrophil independent, ischaemic pathogenesis for NSAID gastric ulceration.

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ulceration along the mesenteric margin
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