A NOVEL ROLE FOR HIGH MOBILITY GROUP BOX 1 (HMGB1) IN GASTRO-OESOPHAGEAL REFLUX DISEASE

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Study of oesophageal biopsies from healthy volunteers and GERD patients finds that refluxate exposure triggers release of HMGB1 from epithelial cells, particularly in response to bile salts, with potential implications for inflammatory signaling in GERD.

KEY POINTS

- HMGB1 mRNA and epithelial protein expression were similar between healthy controls and GERD patients, but TLR4+ epithelial cells were significantly higher in erosive reflux disease compared to controls (7.2% versus 1.4%, $p<0.05$).
- Acidic bile salts, but not acid alone, induced HMGB1 translocation from nucleus to cytosol in cultured oesophageal cells, and synthetic refluxate challenge of biopsies led to increased HMGB1 release over 18 hours (586 versus 218 pg/mL/mg, $p<0.05$).
- RAGE was expressed exclusively on CD45+ leukocytes in GERD tissue, with many RAGE+ immune cells observed in close proximity to sensory CGRP+ nerve fibres.
- The authors suggest HMGB1 may contribute to inflammatory pathways in GERD via TLR4 and RAGE signaling and may be of particular interest in PPI-refractory disease.

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