SMALL BOWEL AND NUTRITION SECTION

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PLASMA GHRELIN PARADOXICALLY RISES UPON FEEDING IN ACTIVE CROHN'S DISEASE

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Introduction Ghrelin is an orexigenic hormone. Circulating levels normally rise between episodes of food ingestion and then fall upon eating. Ghrelin may also be a signal to prime the gut for nutrient absorption. An anti-inflammatory role has also been proposed. A single previous report has suggested an increase in plasma fasting acylated ghrelin expression in active inflammatory bowel disease (IBD), with levels returning to normal once the disease is in remission. The postprandial response of ghrelin in intestinal inflammation has never been described.

Methods 21 patients with active Crohn's disease (CD) and 12 healthy controls were recruited. Disease activity was classified through independent histopathological/endoscopic assessments, validated patient questionnaires and biochemical scores. Gut hormone responses to a mixed nutrient test meal were studied using a multiplex ELISA technique (Luminex). Patient symptoms were assessed using a validated visual analogue score (VAS). A subgroup of patients who achieved remission (without surgery or anti-TNF α treatment) was also re-studied later.

Results CD patients with active inflammation displayed a ~6-fold significant reduction in basal appetite parameters as measured by VAS ($p < 0.0001$). The pattern of the postprandial ghrelin response was paradoxically altered in the CD group with a rise noted postprandially. In the control group ghrelin levels displayed the expected drop after the test meal. Consequently, acylated ghrelin levels were higher in the CD group with a 3-fold increase noted in the total ghrelin level after the test meal.

A significant correlation ($p = 0.019$) was noted between the total acetylated ghrelin secreted and the Crohn's Disease Activity Index. A significant correlation also existed between ghrelin secretion and stool frequency ($p = 0.01$), more so in the SB CD group ($p = 0.004$). There was a significant negative correlation noted between the abdominal pain scores in the visual analogue score specifically in the SB CD group ($p = 0.0008$).

After treatment of CD, the VAS reverted to those of normal controls and ghrelin responses to the test meal now showed a postprandial fall, as expected physiologically.

Conclusion Ghrelin levels show an unexpected rise in response to a test meal in ileal CD. Ghrelin is not expressed in the healthy distal small bowel. It is now necessary to explore whether inflammation induces its expression there, or whether gastric secretion of ghrelin is somehow amplified at a distance. In intestinal inflammation, it may be that ghrelin assumes more its secondary role, that of an anti-inflammatory peptide. This plays a protective role when the antigen load of a meal is presented to the inflamed gut. The changes noted after treatment tend to corroborate this. Its postprandial response may also present a useful novel biomarker of disease activity.

Competing interests None.

Keywords feeding, ghrelin.