Physiology of HCL secretion & peptic ulcer in stomach.

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Introduction

Gastric acid, often known as stomach acid or gastric juice, is a digestive fluid produced by the stomach lining. Gastric acid, which has a pH of 1 to 3, aids protein digestion by activating digestive enzymes, which work together to break down lengthy chains of amino acids. GA is controlled in feedback systems to boost production when it is required, such as after a meal. Bicarbonate, a base, is produced by other cells in the stomach to buffer the fluid and maintain a constant pH. These cells also create mucus, a sticky barrier that protects the stomach from gastric acid. To neutralise gastric acid going into the digestive tract, the pancreas creates enormous amounts of bicarbonate and secretes it through the pancreatic duct to the duodenum.

Peptic ulcer disease (PUD) is a rupture in the stomach's inner lining, the first section of the small intestine, or the lower oesophagus in some cases. A gastric ulcer is one that occurs in the stomach, while a duodenal ulcer occurs in the first part of the intestines. The most typical symptoms of a duodenal ulcer are upper abdomen pain that worsens with eating and waking up with upper abdominal pain. A searing or dull aching is frequently described as the agony. Belching, vomiting, weight loss and a lack of appetite are some of the other symptoms. Bleeding, perforation, and stomach blockage are all possible complications. In as many as 15% of instances, bleeding occurs.

Membrane and protein recycling

Massive membrane modifications are required for stimulation of the gastric parietal cell, as H(+)-pumps from the cytoplasmic tubulovesicle domain are recruited into the apical plasma membrane region. The recycling of membrane pools, which occurs as a result of the fusion and fission processes that occur in response to HCl secretion stimulation and inhibition, also involves highly selective protein incorporation and segregation events. This paper describes a number of proteins found in the apical plasma membrane of maximally stimulated parietal cells, classifying them as either permanent resident proteins of the apical membrane or transient proteins that move into and out of the apical membrane as the cell progresses through the secretory cycle [1].

Protein pump that migrate between tubulovesicles, such as the 94 kDa catalytic alpha-subunit of the H+K(+)-ATPase and its newly identified beta-subunit glycoprotein, are a good example of temporary connection with the apical membrane. Actin and an 80-kDa phosphoprotein known variably as 80 K, ezrin, p81, and cytovillin stay linked with the apical plasma membrane during rest and secretion, and whose phosphorylation is stimulated by the histamine/cAMP route of parietal cell stimulation. A 120-kDa cytosolic protein appears to have protein kinase activity and gets linked with the apical plasma membrane after stimulation. It's worth noting that the identification, location, and characterisation of the K+ and Cl- transport proteins involved in net HCl secretion are urgently needed [1].

Stress ulcers are numerous, superficial erosions that mostly affect the stomach's fundus and body. They commonly occur in people with peritonitis and other chronic medical illnesses, and they arise after shock, infection, or trauma. Reactivation of chronic duodenal or gastric ulcers should be distinguished from stress ulcers. Drug-induced gastritis or Cushing's ulcer after a brain injury. Digestive symptoms are rarely present, bleeding is the most prevalent symptom, and perforation and blockage are uncommon. The presence of luminal acid and ischemia is required for the development of a stress ulcer, and the breakdown of the gastric mucosal barrier caused by refluxed duodenal material may contribute to pathogenesis. Endoscopy is the gold standard for diagnosing bleeding lesions, however angiography should be done if endoscopy fails to reveal them [2].

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Conclusion

The presence of luminal acid and ischemia is required for the development of a stress ulcer, and the breakdown of the gastric mucosal barrier caused by refluxed duodenal material may contribute to pathogenesis. Endoscopy is the gold standard for diagnosing bleeding lesions, however angiography should be done if endoscopy fails to reveal them.

References

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