

## ABSTRACT

Key words:  $\beta_2$ -adrenergic agonist  
gastric acid  
plasma  $\text{HCO}_3^-$   
gastric mucus

An experimental study has been done to investigate the role of  $\beta_2$ -adrenoceptor agonists (salbutamol, salmeterol and fenoterol) on the gastric acidity and the protective factors of gastric mucosa. The role of the sympathetic nervous system on the pathophysiology of peptic ulcer is not completely understood.

In this study which has been done in vivo and in vitro gastric acid secretion was measured besides the level of plasma  $\text{HCO}_3^-$  and thickness of mucus layer on the gastric wall as indicators of gastric defence mechanism.

The study of gastric acidity in vivo was carried out on rats using the Shay method; in vitro it was done on isolated fundi of the stomach. The plasma  $\text{HCO}_3^-$  level was measured in the blood aspirated directly from the rat's hearts. The thickness of the mucus was studied using a simple method developed by Kerss, Allen, Garner (1982).

The result of the study showed a significant role of  $\beta_2$ -adrenoceptor agonist in inhibiting the basal state as well as histamine and carbamylcholine-stimulated acid secretion respectively.  $\beta_2$ -adrenoceptor agonist also increased the plasma  $\text{HCO}_3^-$  level and the thickness of the mucus gel which adheres to the surface of the gastric

epithelial cells, both in basal condition as well as after histamine or carbamylcholine stimulation.

There is no significant difference in the effects of inhibition of gastric acidity, increased plasma  $\text{HCO}_3^-$  and the thickness of gastric gel mucus layer by the three different  $\beta_2$ -adrenergic agonists. Furthermore administration of propranolol abolish the effect of  $\beta_2$ -adrenoceptor agonist on gastric acidity, plasma  $\text{HCO}_3^-$  level and gastric gel mucus thickness.

Peptic ulcer disease arises when the normal mucosal defence factors are impaired or are overwhelmed by aggressive luminal factors such as acid and pepsin. The mucus gel provides an unstirred water layer, which slows inward diffusion of  $\text{H}^+$  toward the mucosa. Bicarbonate which is secreted by surface epithelial cells throughout the stomach converts  $\text{H}^+$  penetrating the mucus layer to  $\text{CO}_2$  and  $\text{H}_2\text{O}$ . Both the mucus layer and secretion of  $\text{HCO}_3^-$  serves as a protection of the surface epithelium against injurious acid.

From this study we conclude that the sympathetic nervous system via  $\beta_2$ -adrenergic receptors might play an important inhibitory role in the development of peptic ulcer disease by decreasing gastric acidity, increasing plasma  $\text{HCO}_3^-$  level and the thickness of the gastric mucus gel. More studies are needed to confirm this observations.