Electrolytes & [H+] Disturbances in Peptic Ulcer Patients

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Abstract

In the body even a very small pH changes are important and result in change in body physiology. We describe in our study the presence of alteration in electrolytes and acid-base status in patients with peptic ulcer disease. The study was carried out over a period of three years. 273 patients with peptic ulcer disease diagnosed on endoscopy, of these 228 duodenal ulcer and 45 gastric ulcer, in addition 84 subjects were considered as a healthy control.

20 ml of gastric juice obtained for analysis and 5 ml blood sample were taken and pH determination and [H+] estimated by electric sensitive method including the use of electronic sensitive pH – meter. The pH – electrode was initially calibrated in buffer solution of pH 7 ( Ian 1969 ). Also the total acidity of gastric effluents were estimated by titration to pH 7 with 0.1 NaOH. The K+ and Na+ concentrations determined by using flame photometric method ( Sood 1987 ) The results of the study show that DU patients had significantly lower gastric secretion pH and higher H+ concentration in the blood than normal control subjects and total acidity of gastric secretion also was higher than control.

Introduction

During discovery of gastric acid, four phases of discovery led to general acceptance that the stomach secretes HCL and these include firstly the appreciation that gastric contents might be acid, secondly long arguments established whether, this acid was secreted by the stomach or whether it is produced by fermentation of food, and then qualitative determination of acid and followed by quantitative measurement of basal and maximal acid output. (1) There are at present two contenders: a chemical enzyme inhibitors associated with the NSAIDs induced ulcers & the role produced by H. pylori infection as a second contender.

During the 6th Century BC The Babylonian
Talmud notes the effect of saliva and churning action of the stomach in digestion. This means that the GIT problems are always present throughout human history. The problem of gastric acid secretion was demonstrated by William Prout in 1824, he demonstrated the presence of hydrochloric acid in gastric juice, and in 1915 B. W. Sippy advocates an acid-neutralization regimen for treatment of peptic ulcer. In 1972 A. Allen and associates begin their studies on the biochemistry of gastric mucus and in 1977 G. Flemstrom and associates elucidate the kinetics and mechanism of gastric bicarbonates production. The concept of cytoprotection against development of ulcer suggested earlier by T. K. Chaudhur & E. D. Jacobson (1978) and at the same period J. I. Rotter et al. proposed that elevated serum pepsinogen serves as a marker for predisposition to DU. In 1983 J. R. Warren & B.J. Marshall rediscovers H. pylori and its association with antral gastritis and peptic ulcer. (2)

It is known fact that peptic ulcer disease refers to the underlying tendency to develop mucosal ulcer at sites that exposed to acid & pepsin and compared with age-matched controls patients with DU secretes more acid during the day & night and exogenous and endogenous factors alter the lines of mucosal defense, allowing back diffusion of H+ ions and subsequent injury of epithelial cells. (3)

Acid–Base balance (ABB) modulates the gut electrolytes transport both in vivo & in vitro. Changes C/W metabolic acidosis are potent stimulators of electroneural sodium chloride absorption, whereas metabolic alkalosis inhibits this transport process. (4, 5) Studies involving measurements of intracellular pH suggest that intracellular bicarbonate concentration may modulate basal chloride secretion and intracellular pH and P CO2 may alter Na+:H+ exchange and this might contribute to alteration in H+ ions secretion in conditions of (ABB) disturbances. The blood H+ concentration is maintained within a tight limits in health. Normal level between 35 – 45 nmol/L, and now it is better to describe H+ concentration than to say the pH and this is maintained about 40 nmol/L by the normal pattern of both renal & respiratory systems function.(6,7)

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Patients and Methods

The study was carried out over a period of three years. 273 patients with peptic ulcer disease diagnosed on endoscopy, of these 228 duodenal ulcer and 45 gastric ulcer, in addition 84 subjects were considered as a healthy control.

20 ml of gastric juice obtained for analysis and 5 ml blood sample were taken for pH determination and [H+] estimated by electric sensitive method including the use of electronic sensitive pH – meter. The pH – electrode was initially calibrated in buffer solution of pH 7 (Ian 1969) (8). Also the total acidity of gastric effluents were estimated by titration to pH 7 with 0.1 NaOH. The K+ and Na+ concentrations determined by using flame photometric method (Sood 1987) (9). The serum Calcium was estimated by colorimetric microdetermination technique and the Ca – Ocp – Kit was used. (Harold, et al.1980) (10)

Statistical methods were expressed as mean ± SE. The data were analysed using t-test and taking (P value < 0.05) as lowest limit of significance and to determine significant correlation qui square test was used.
The results

The results of the study show that DU patients had significantly lower gastric secretion pH and higher H+ concentration in the blood than normal control subjects and total acidity of gastric secretion also was higher than control. (See table I) We measure the total acidity of gastric secretion in patients with GU and it was lower than control groups. During the course of the study we had noticed that patients with peptic ulcer infected with H. pylori show higher total acidity than patients having no H. pylori infection.

In DU patients, there is significant (P < 0.01) increase in the concentration of gastric secretion of K+ compared with the control and in contrast, there is significant (P < 0.01) decrease in the concentration of gastric secretion of K+ in GU patients compared with control subjects. (see table II) There is non-significant differences (P > 0.05) in the concentrations of serum K+ between peptic ulcer patients & control.

DU patients have significantly (P < 0.01) lower concentration of Na+ in gastric secretion than control, whereas GU patients show higher concentration of gastric secretion Na+ than control. Also during the course of study we noticed H. pylori positive patients show decrease in the concentration of gastric secretion Na+ than normal control subjects and H. pylori negative patients. There was no significant differences in the concentration of serum Na+ in PU patients and control groups.

There was a significantly higher concentration of serum calcium in PU patients than control. (see table III) And see also figures No. 1 to figure No. 5 which demonstrate gastric secretions pH, Na+, K+ and also serum Na+ and serum Calcium ions.

Table 1. Blood H+ concentration in PU patients & control

<table>
<thead>
<tr>
<th>Subjects</th>
<th>H+ nmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>DU +ve Hp</td>
<td>78.67±8</td>
</tr>
<tr>
<td>DU -ve Hp</td>
<td>67±12</td>
</tr>
<tr>
<td>GU +ve Hp( H. Pylori)</td>
<td>30.5±1.2</td>
</tr>
<tr>
<td>GU -ve Hp</td>
<td>28.5±3</td>
</tr>
<tr>
<td>Control</td>
<td>40±5.5</td>
</tr>
<tr>
<td>P value</td>
<td>P &lt; 0.01</td>
</tr>
</tbody>
</table>

Table 2. Shows concentrations of K+, Na+ in gastric secretions of DU patients, GU patients, and control

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Mean and SD in mmol/L of K+ conc.</th>
<th>Mean &amp; SD in mmol/L of Na+ conc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>DU</td>
<td>22±2</td>
<td>64±6</td>
</tr>
<tr>
<td>DU with Hp infection</td>
<td>25±3</td>
<td>60±7</td>
</tr>
<tr>
<td>GU</td>
<td>11±3</td>
<td>100.5±7</td>
</tr>
<tr>
<td>GU with Hp infection</td>
<td>13±1</td>
<td>95±6</td>
</tr>
<tr>
<td>Control</td>
<td>16±3</td>
<td>90.5±8</td>
</tr>
<tr>
<td>P value</td>
<td>(P &lt; 0.05)</td>
<td>(P &lt; 0.05)</td>
</tr>
</tbody>
</table>

Table (3) shows the serum Calcium concentration in peptic ulcer patients & control groups:

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Serum Calcium mg/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>DU</td>
<td>11.21±1</td>
</tr>
<tr>
<td>GU</td>
<td>10.91±1.2</td>
</tr>
<tr>
<td>Control</td>
<td>9.52±0.7</td>
</tr>
<tr>
<td>P value</td>
<td>(P &lt; 0.01)</td>
</tr>
</tbody>
</table>
Discussion & Conclusion

Patients with DU disease show disturbances in electrolytes in the gastric secretion, which also associated with with excess HCL secretion. In general Na+ concentration in gastric juice is inversely proportional to the H+ concentration in gastric secretion and this might reflect the fact that electrolytes are involved in exchanges of H+ , but not in the integrity of gastric barrier, although Davenport showed that measurement of electrolytes exchange across the mucosa gives a reliable estimation of integrity of gastric mucosal barrier . (11)

DU patients show significantly lower concentration of Na+ in gastric secretion than control , whereas GU patients show higher values . (12)

The elevated serum calcium level is consistent with other several studies (13), and it had been suggested that calcium has a role in the pathogenesis of PU through the obligatory effect in stimulation of gastric acid secretion through , gastrin, histamine and cyclic AMP & these might be an important steps in pathogenesis of peptic ulcer and mucosal injury . (14,15,16,17) Also the effect of elevated calcium in PU disease might be attributed to hormonal effect . (18,19,20)

In one study which enrolled 74 patients with DU , 28 patients with chronic gastritis and 16 healthy control , the secretion was studied initially , then in administration of ranitidine and in stimulation of hydrochloric acid . The result of this study showed that DU patients demonstrated a significant reduction of gastric secretion of HCO3− in basal & stimulated phases and three fold increase in the proportion of alkaline / acid secretion and there was also trend toward acidosis , this consistent with our study which suggest there is high concentration of H+ in the blood in patient with PU . In addition to acidosis , HCO3− deficiency is an important element in pathogenesis of DU . (21) Gastric mucosal acid – base balance play a significant role in protection against development of mucosal injuries and if there is loss of the protective alkaline efflux & exposure to further intraluminal acid and any barrier- breaking agents result in intracellular acidosis . (22)

In another study which concludes that acidosis contributes to exacerbation of peptic ulcer & giving Ranitidine to these patients restored acid- base balances . (23) Our study goes with the conclusion of Malov who said that acid – base balance & gastric secretion of HCO3− are related and acidosis in peptic ulcer patients lead to reduction of HCO3− production and intensification of of hydrochloric acid secretion . (24)

In Conclusion : PU disease is associated with disturbances in Electrolytes and Hydrogen ions concentration in the blood & gastric secretion . Also in DU patients , there is metabolic acidosis which leads to disturbance of cytoprotective mechanism of gastro-duodenal mucosa and mucosal production of HCO3−.

References

24. Malov IuS , Ivashkina T. G. , relationof acid-bas balance& gastric secretion of Hydrogen ion, Carbonates ions in patients with PU . Ter Arkh , 2001 ; 73 ( 2 ) :6-10 .Figures No. 1 to 5 :