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Chemical Analysis of Gastric Juice in Duodenal Ulcer Patients Infected with *Helicobacter Pylori*

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ABSTRACT

The research was concerned with chemical analysis of the gastric juice in duodenal ulcer patients caused by *Helicobacter pylori* infection. The study included (25) patients (13 males and 12 females) attending endoscopy unit of Salam General Hospital where gastric juice was taken. Also gastric juice from (25) normal healthy subjects (13 from males and 12 females) as control were collected for chemical analysis. The chemical analysis of gastric juice included measurement of electrolytes (Na^+, K^+, Ca^{+2} and Cl^-) lactate, glucose, total lipids, iron in addition to conductivity and viscosity. The results of statistical analysis predicted significant differences in the levels of sodium, chloride, iron, lactate, glucose and conductivity in gastric juice of duodenal ulcer male patients infected with *H. pylori* in comparison to control male. However, potassium, calcium, total lipids and viscosity showed non significant difference. Females patients, however, showed significant differences for sodium, iron, calcium, lactate, glucose, total lipids and viscosity while potassium, chloride and conductivity showed non significant differences in comparison with healthy females.

التحليل الكيميائي لعصارة معدة المرضى المصابين بقرحة الاثني عشر المسببة عن البكتريا الملوية البوابية

الملخص

تضمن البحث التحليل الكيميائي لعصارة معدة المرضى المصابين بقرحة العفج (الاثني عشر) المسببة عن البكتريا الملوية البوابية. حيث شملت الدراسة سحب عصارة معدة (25) مراجعا (13 ذكور و 12 اناث) لوحـدة الناظور في مستشفى السلام العام. كذلك جمعت (25) عينة من عصارة المعدة (13 ذكور و 12 اناث) لاشخاص اصحاء كمجموعة ضابطة. شمل التحليل الكيميائي لعصارة المعدة قياس الالكتروليتـات (صـوديوم، بوتاسـيوم، كالسيوم والكلوريد) واللاكتيت والكلوكوز والدهون الكلية والحديد بالاضافة الى قيـاس التوصـيلية واللزوجـة. اظهرت نتائج التحليل الاحصائي وجود اختلافات معنوية في مستويات الصوديوم، الكلور، الحديـد، اللاكتيـت، الكلوكوز والتوصيلية في عصارة معدة الذكور المصابين بقرحة الاثني عشر المسببة عن البكتريا الملوية البوابية مقارنة بالذكور الاصحاء. واظهر كل من البوتاسيوم والكالسيوم والدهون الكلية واللزوجة عدم وجود فرق معنوي.

اظهرت النتائج ان الاناث المصابات بقرحة الاثني عشر وجود اختلافات معنوية في الصوديوم، الحديد، الكالسيوم، اللاكتيت، الكلوكوز، الدهون الكلية واللازوجة، وعدم وجود فرق معنوي في مستوى البوتاسيوم والكلوريد والتوصيلية مقارنة بالاناث الاصحاء.

INTRODUCTION

Peptic ulcer is a result of self-digestion resulting from an excess of autopeptic power of gastric juice over the defensive power of gastric mucosa (Samloff, 1989; Werther, 2000). Patients with duodenal ulcer have a higher acid outputs than the controls (Stockbruegger, 1984). About (30%) have higher basal acid secretion probably because of a large parietal cell mass. Nacturnal rates of acid secretion are elevated and there is a higher peak secretion of acids (Richardson, 1986). Many acid secretory abnormalities have been described in duodenal ulcer patients. The reason for this altered secretory process is unclear yet but *H. pylori* infection may contribute to this finding (Kasper et al., 2005).

The secretions of the digestive tract are elaborated by specialized glands. They provide mixtures of varying electrolytes composition and contain enzymes, proenzymes and other substances. The electrolytes composition of parietal gastric juice and gastric mucus involved (Na^+, K^+, Ca^{++}, Cl^- and HCO_3^-). It can be noted that these secretions are virtually isotonic with plasma (Smith et al., 1983). D-lactate, a metabolic product of some bacteria, may be produced in excess if there is overgrowth of such organisms in the human gut. This can occur when a large amount of the small gut is resected and the remaining gut is colonized by D-lactate producing organisms (Walmsley and White, 1988).

This study was designed to asses the biochemical changes in gastric juice sodium, potassium, calcium, chloride and the presence of iron, glucose, total lipids and lactate in duodenal ulcer patients with *H. pylori* infection.

MATERIAL AND METHODS

Patients are enrolled in the present study to the gastroendoscopy unit in Al-Salam General Teaching Hospital in the fasting state (no food or liquid for at least 12 hours).

Collection of gastric juice sample:

Gastric juice from (25) patients were collected. In these patients, gastroscopy confirmed the presence of duodenal ulceration and *H. pylori* infection. The juice (5-10)ml was collected through the suction line of endoscope immediately after the line reached the stomach. The sample was then centrifuged at (3000 xg) for (10) minutes to remove mucous (Tamura et al., 1997; Tokushima et al., 1998). Gastric juice sample from (25) individuals without dyspeptic symptoms and normal at the endoscopic examination were collected and considered as control.

Methods:

Sodium, potassium and calcium were determined using blood gas and electrolytes analyzer (the eschweiler COMBISYS H) is a microprocessor controlled automatic analysis system for quantitative sample of whole blood or serum present in the laboratory of Al-Salam Hospital.

K^+ measuring range = 2 – 10 mmol/L.
Na^+ measuring range = 100 – 200 mmol/L.
Ca^+ measuring range = 0.5 – 2.0 mmol/L.

Glucose was assayed using manufactured kit by Diamond, Jordan. Lactate concentration was measured by an enzymatic method (Varley and Gowen, 1980), while total lipids was determined spectrophotometrically (Chabrol and Chardonnet, 1937). Chloride ion concentration was determined spectrophotometrically at 348 nm (Snell, 1965). Iron concentration was determined using manufactured kit by SyrBio (Ceriotti and Ceriotti, 1980). Conductivity was measured using conductmeter, while viscosity of gastric juice was measured using viscometer (ASTM, 1973).

RESULTS AND DISCUSSION

The results in Table (1) showed significant increase of sodium concentration in gastric juice of duodenal ulcer patients (males and females) infected with *H. pylori* with comparison to control. This might be due to a pancreatic juice exits from the cinar region travels down the ducts, the initial concentration of major electrolytes Na^+, K^+, Cl^- and HCO_3^- reflect. That of the extra cellular fluid. Upon stimulation of the duct cells as ordinarily occurs during and immediately after a meal, there are specific changes in the electrolyte composition of the pancreatic juice emptied into the duodenum (Sperelakis, 1993) or due to increased bile regurgitation into the stomach occurs in peptic ulcer disease (Werther, 2000). While potassium concentration showed a non significant difference between patients (males and females) and control as shown in Table (1), because all gastrointestinal secretions contain potassium (Kravis et al., 1993); any patient with fluid loss from the gastrointestinal tract should be considered a candidate for hypokalemia due to vomiting and aspiration of gut secretion (Laker, 1996). On the other hand a significant increase of chloride concentration was observed on gastric juice of duodenal ulcer males patients infected with *H. pylori*, this is due to that *H. pylori* infection in patients leading to increased release of gastric juice from antral G cells so the formation of gastric acid and hyperchlorhydria is one of the physiopathological implications of gastric (Huang, 2000), while the concentration of chloride in the gastric juice of females patients give nonsignificant increase in comparison with the control for unknown reason.

Table 1 : The chemical analysis of electrolytes on gastric juice of duodenal ulcer patients infected with *H. pylori*.

Parameters mmol/L	**Males (13) (Mean ± SD)**		**Females (12) (Mean ± SD)**	
	Patients	**Control**	**Patients**	**Control**
Sodium	***483.69 ± 275.3	41.15 ± 13.1	*** 373.75 ± 248.2	52.66 ± 29.1
Potassium	14.66 ± 6.2	19.53 ± 9.2	14.06 ± 2.5	17.29 ± 11.1
Calcium	0.39 ± 0.31	0.52 ± 0.5	*0.35 ± 0.2	0.74 ± 0.4
Chloride	**148.92 ± 18.1	122.15 ± 32.3	135.16 ± 11.0	137.33 ± 31.6

* The results are significantly at $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

In this study a non significant and significant differences were found between calcium concentration in gastric juice of duodenal ulcer patients (males and females) respectively according to the control Table (1). This is due to secretion of specialized fluids is generally accomplished by epithelial cells aligned in columnar fashion, bathed by interstitial fluid of plasma on one side and fluid of different composition on the opposite side. Presence of Ca^{++} in gastric juice is due to reflux from pancreatic juice to duodenum (Smith et al., 1983).

The results obtained in Table (2), a significant increase of lactate in gastric juice of duodenal ulcer patients infected with *H. pylori* in comparison control. This is due to bacterial action when food is retained in the stomach for long periods (> 6h) (Tietz. 1996) or due to a large amount of their small intestine secretion which occur after carbohydrate meal which is not properly digested and reaches bacteria in sufficient quantities to be metabolized to lactic acid (Walmsley and White, 1988).

Also the results obtained in Table (2), a significant decrease of glucose in gastric juice of duodenal ulcer patients (males and females) infected with *H. pylori* in comparison to control. This is due to the vagal stimulation of gastric secretion can be tested by inducing hypoglycaemia with insulin in duodenal ulceration (Zilva et al., 1988; Barakat, 1991). Yet, total lipids give a significant results in females and non significant results in males in comparison to control. The stomach contains a gastric lipase capable of hydrolyzing triacyl glycerols of short and longer chain length. However, the lingual lipase can continue its activity at the low pH of the stomach, where because of the retention time of (2-4) hours, about 30% of dietary triacyl glycerol may be digested (Murray et al., 1993).

Table 2 : The concentrations of lactate, glucose total lipids and iron in gastric juice of duodenal ulcer patients infected with *H. pylori*

Parameters	**Males (13) (Mean ± SD)**		**Females (12) (Mean ± SD)**	
	Patients	**Control**	**Patients**	**Control**
Lactate (mmol/L)	***3.01 ± 0.3	1.83 ± 0.2	***2.65 ± 0.2	1.71 ± 0.17
Glucose (mmol/L)	***0.088 ± 0.06	0.456 ± 0.21	**0.198 ± 0.24	0.540 ± 0.34
Total lipids (mg/dl)	46.615 ± 20.8	51.254 ± 21.1	***58.66 ± 18.8	27.016 ± 14.83
Iron (mg/dl)	**7.17 ± 4.0	3.25 ± 2.1	**7.69 ± 3.2	3.22 ± 8.2

** The results are significantly at $p \leq 0.01$; *** $p \leq 0.001$

Also this study indicated a significantly higher concentration of iron in gastric juice of duodenal ulcer patients (males and females) infected with *H. pylori* than in controls, because *H. pylori* infection reduced the release of gastric epidermal growth factor and may facilitate the development of mucosal damage, it also plays a major role in ulcer formation (Huang, 2000) or due to accidental trauma from the gastric tube (Tietz, 1996).

The study indicated a significant increase in male patients and non significant difference between females patients and control in conductivity and opposite results of viscosity as shown in Figure (1). This probably due to increased hypersecretion of gastric acid (Zilva, 1988). It is known that plasma is the source of Cl^- ion, it is pumped by an active transport process into lumen by the gastric mucosal cell. This pump is electrogenic and gives rise to a potential difference such as that the luminal surface of the gastric cell is negative with respect to the serosal surface of the cell. The exchange is driven by energy mode available by hydrolysis of ATP. The ATPase involved is stimulated by K^+, Mg^{++} but is insensitive to Na^+. Thus, it differ from the usual Na^+, K^+, ATPase or Na^+ pump (Smith et al., 1983).

Also, in this study a non significant differences between chemical analysis in gastric juice of duodenal ulcer patients males and females is observed except that lactate and chloride concentrations give a significant differences between subjects, as shown in Table (3).

Table 3 : The statistical analysis between parameters in gastric juice of duodenal ulcer patients (males and females)

Parameters	**Sex**	
	Male (13) Mean ± SD	**female (12) Mean ± SD**
Sodium (mmol/L)	483.69 ± 275.3	373.75 ± 248.2
Potassium (mmol/L)	14.66 ± 6.2	14.06 ± 2.5
Calcium (mmol/L)	0.39 ± 0.31	0.35 ± 0.2
Chloride (mmol/L)	*148.92 ± 18.1	135.16 ± 11.0
Lactate (mmol/L)	*3.01 ± 0.3	7.69 ± 3.2
Glucose (mmol/L)	8.846 ± 6.13	0.195 ± 0.24
Total lipids (mg/dl)	46.62 ± 20.84	58.67 ± 18.84
Iron (mg/dl)	7.17 ± 4.0	7.69 ± 3.2
Conductivity (ms)	9.94 ± 4.5	7.81 ± 5.4
Viscosity (poise)	0.85 ± 0.7	0.83 ± 0.0

* Means ± SD significantly differences at $p \leq 0.05$

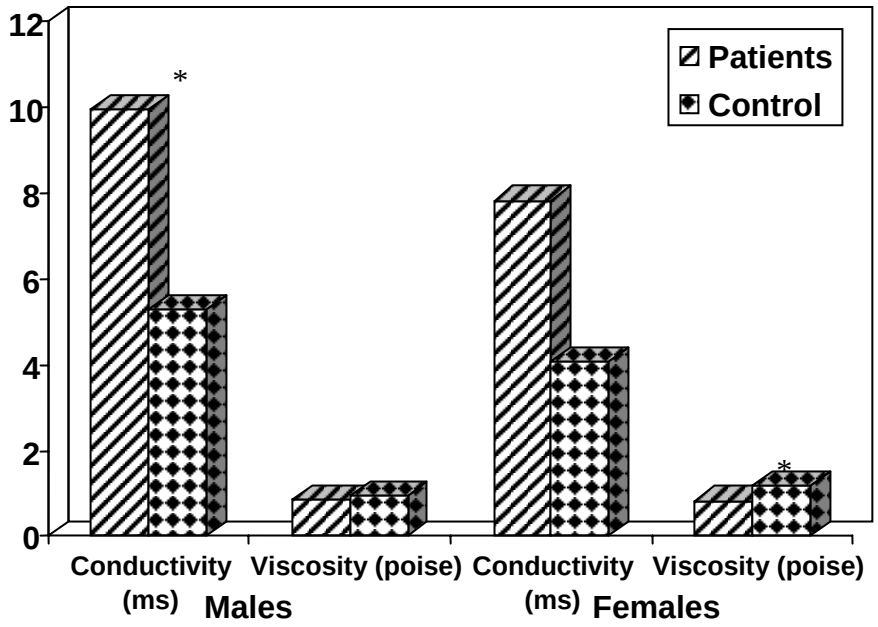

Figure 1 : The conductivity and viscosity of gastric juice in duodenal ulcer patients infected with *H. pylori*.
* significant at $p \leq 0.05$

REFERENCES

Barakat, M., 1991. Surgery of Digestion, Part 1, College of Medicine, University of Halab, 145 p.

Ceriotti, F. and Ceriott, G., 1980. Improved Direct Specific Determination of Serum Iron. Clin. Chem. Vol. 26, No. 2, pp. 327-331.

Chabrol and Chardonnet, 1937. Cited by Gelson Tono and Philip G. Ackrmann (1975), Practical Clinical Chemistry, Little, Brown and Company, Boston.

Huang, X.Q., 2000. *Helicobacter pylori* Infection and Gastrointestinal Hormones; a Review, World J. Gastroenterology, 6(6): pp.783-788.

Kasper, D.L., Fauci, A.S., Longo, D.L., Braunwald, E., Hauser, S.L. and Jameson, J.L., 2005. Harrison's Principles of Internal Medicine, 16th ed., Medical Publishing Division, New York, San Francisco, Washington DC, Auckland Bogota, Vol. 1-4, 1649 p.

Kravis, T.C., Warner, C.G. and Jacobs, L.M., 1993. Emergency Medicine A Comprehensive Review, 3rd ed., Raven Press, New York, 294 p.

Laker, M.F., 1996. Clinical Biochemistry for Medical Students, W.B. Saunders Company Limited, London, Philadelphia, Toronto, Sydney, Tokyo, pp.90-97.

Murray, R.K., Mayes, P.A., Granner, D.K. and Rodwell, V.W., 1993. Harper's Biochemistry, 22nd ed., Middle Eqst Edition, Librairiedu Liban, Appleton and Lange.

Richardson, F.M., 1986. Total 24-Hour Hastric Secretion in Patients with Duodenal Ulcer Comparison with Normal Subjects and Effects of Cimetidine and Parietal Cell Vagotomy, Gastroenterology, 90: pp.540-544.

Samloff, I.M., 1989. Peptic Ulcer: The Many Proteinases of Aggression. Gastroenterology, 96: pp.586-595.

Smith, E.L., Hill, R.L., Lehman, I.R., Lefkowitz, R.J., Handler, P. and White, A., 1983. Principles of Biochemistry, Mammalian Biochemistry, 7th ed., McGraw-Hill Book Company.

Snell, F.D., 1965. Photometric and Fluorometric Methods of Analysis of Nonmetals, John Wiley and Sons, New York, Chichester, 250 p.

Sperelakis, N. and Banks, R.O., 1996. Essentials of Physiology, 2nd ed., Little, Brown and Company, USA.

Standard Method of test for kinematic viscosity of transparent and opaque liquids (and the calculation of dynamic viscosity), 1973. Annual Book of ASTM standards Part 18m p: D445-72 Revision Issued Annually (Annive Rsary).

Stockbruegger, R., 1984. Ulcer. Boehringer Ingelhcim International GmbH.

Tamura, H., Tokushima, H., Murakawa, M., Matsumura, O., Itoyama, S., Sekin, S., Hirose, H., Mitarai, T. and Isoda, K., 1997. Eradication of *Helicobacter pylori* in Patients with End-Stage Renal Disease Under Dialysis Treatment, Am. J. Kidney Dis., 29(1): pp.86-90.

Tietz, N.W., 1996. Textbook of Clinical Chemistry, W.B. Saunders Company, USA, A Division of Harcourt Brace and Company, Philadelphia.

Tokushima, H., Tamura, H., Murakawa, M., Matsumura, O., Itakura, Y., Itoyama, S., Mitarai, T. and Isoda, K., 1998. Eradication of *Helicobacter pylori* Restores Elevation of Serum Gastric Concentrations in Patients with End-Stage Renal Disease, Intern. Med., 37(5): pp.435-439.

Varley, H. and Gowenlock, A.H., 1980. Practical Clinical Biochemistry, 5th ed., William Heinemann Medical Books Ltd., London, pp.397-398, 819-820.

Walmsley, R.N. and White, G.H., 1988. A Guide to Diagnostic Clinical Chemistry, 2nd ed., Blackwell Scientific Publications.

Werther, J.L., 2000. The Gastric Mucosal Barrier, The Mount Sinai Journal of Medicine, 67(1): pp.41-54.

Zilva, J.F., Pannall, P.R. and Mayne, P.D., 1988. Clinical Chemistry in Diagnosis and Treatment, 5th ed., Edward Arnold, A Division of Holdder and Stoughton, London.