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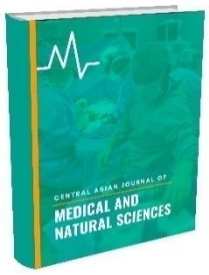

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Evolution Levels of Pancreatic Amylase and Plasma Ammonia in Patients with Gastric Ulcer Disease

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Abstract: The purpose of this research was to evolute levels of pancreatic amylase and plasma ammonia in patients with gastric ulcer from Al Fayhaa General Hospital. Peripheral blood samples of all subjects were collected for Serum amylase and plasma ammonia estimation. The results showed that high prevalence of gastric ulcer 40(53%) were in age group over 65 years and there was significant increase in Ammonia in group 3 and group 4 of (40-65) and over 65 years respectively. In addition to significant increase in Amylase in group 4 as compared with the control group.

Key words: gastric ulcer, plasma ammonia, Serum amylase.

Introduction

Peptic ulcer disease is among several upper gastrointestinal tract disorders that are partially caused by gastric acid (1). Duodenal and gastric ulcers develop when there are breaks in the duodenal and gastric mucosa and both are related to the corrosive effect of hydrochloric acid and pepsin on the upper gastrointestinal tract's mucosa (2). The hormone Cholecystokinin is also released into the bloodstream during digestion. The hormone, CCK is responsible for the secretion of the pancreatic enzymes and bile by the pancreas and liver, respectively, into the small intestine. This hormone is responsible for the contraction of the stomach, which makes the person feel full, thereby enabling appetite control. On the other hand, wheat germ lowers CCK levels; hence, it must be avoided (3). The pancreatic enzymes help in the breakdown of fats, proteins and carbohydrates. Though the digestion of proteins initiates in the stomach, it is completed in the small intestine (4). The enzymes in the small intestine enable the breakdown of the food before its absorption in the body (5).

Methods

The study was taken 100 subjects with symptoms of gastric ulcer, with age range between 25 and over 65 years, patients were selected from Al Fayhaa General Hospital and were exposure for endoscopy to detect gastric ulcer. 25 subjects were healthy (Group 1: control group with age range 25-over 65 year), while 75 with gastric ulcer and classified according to age into three groups Group 2 (25-40) year, Group 3 (40-65) year and Group 4 (over 65 year). Peripheral blood samples of all subjects were collected. The collected samples will be transferred to 5ml tubes, and kept in EDTA-coated tubes for Serum amylase and plasma ammonia estimation. Gastric ulcer in all patients was due to NSAID using.

RESULTS

Distribution ages of patients with gastric ulcer. After the analysis of 75 patients, about 15 (20%) were in (25-40) age group, while about 20 (27%) were in (40-65) age group and 40(53%) were in age group over 65 years as shown in (Table. 1).

Table 1: Distribution age of patient with gastric ulcer

Frequency (%)	Age group (year)
15(20)	25-40
20(27)	40-65
40(53)	Over 65
75(100)	

The result exposed that significant increase in Ammonia in group 3 and group 4, and significant increase in Amylase in group 4 as compared with the control group as shown in (Table. 2).

Table 2: Effect of gastric ulcer on serum amylase and plasma ammonia.

Variables	Serum amylase (U/L)	Plasma ammonia (Umol/L)
Group 1 (Control)	88.50 ± 1.79	45.80 ± 2.11
Group 2	76.5 ± 1.87	51.80 ± 1.91
Group 3	96.0 ± 2.81	63.19 ± 2.21*
Group 4	126.0 ± 4.11*	86.89 ± 2.75*

Discussion

There was significant increase of serum amylase in group 3and group 4 with ages 40 to over than 65 years as compared to the Group 1 (control group), and there was significant increase in Ammonia in group 3 and group 4 of (40-65) and over 65 years respectively. The significant increase in serum amylase in group 4 May be due to physiological effect in pancreatic diseases caused by gastric ulcer. This effect was attributed to the increased gastrin and reduced somatostatin release in these subjects because of direct action of gastric ulcer and its poisons on the D-cells and G-cells in the gastric mucosa. This increase in Amylase activity might be also related to the raise of ammonia (6). The hormone Cholecystokinin is also released into the bloodstream during digestion. The hormone, CCK is responsible for the secretion of the pancreatic enzymes and bile by the pancreas and liver, respectively, into the small intestine (7). This hormone is responsible for the contraction of the stomach, which makes the person feel full, thereby enabling appetite control. The pancreatic enzymes help in the breakdown of fats, proteins and carbohydrates (8). Though the digestion of proteins initiates in the stomach, it is completed in the small intestine. The enzymes in the small intestine enable the breakdown of the food before its absorption in the body. The colon, or large intestine, absorbs the maximal amount of water. Hence, it requires a lot of fiber for adding bulk to its content. The insoluble fiber in the food acts as a sponge, which soaks the liquid and helps the passage of the food through the digestive tract (9). On the other hand, soluble fiber binds to the toxins in the digestive tract, softens the stool and helps their smooth excretion. Along with the classification of fiber as soluble or insoluble, every individual possesses a different fiber composition. Though the body cannot digest fiber, the gut microflora can easily digest it. The gut microflora depends on the blood group of the person (10). Ingestion of a wrong type of fiber can lead to bloating and flatulence.

Reference

1. Narayanan M, Reddy KM, Marsicano E. Peptic Ulcer Disease and *Helicobacter pylori* infection. Mo Med. 2018 May-Jun;115(3):219-224.
2. Lanas Á, Carrera-Lasfuentes P, Arguedas Y, García S, Bujanda L, Calvet X, Ponce J, Perez-Aísa Á, Castro M, Muñoz M, Sostres C, García-Rodríguez LA. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants. Clin Gastroenterol Hepatol. 2015 May;13(5):906-12.e2.
3. Huang JQ, Sridhar S, Hunt RH. Role of Helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: a meta-analysis. Lancet. 2002 Jan 05;359(9300):14-22.
4. Snowden FM. Emerging and reemerging diseases: a historical perspective. Immunol Rev. 2008 Oct;225(1):9-26.
5. Lanas A, Chan FKL. Peptic ulcer disease. Lancet. 2017 Aug 05;390(10094):613-624.
6. ASGE Standards of Practice Committee. Banerjee S, Cash BD, Dominitz JA, Baron TH, Anderson MA, Ben-Menachem T, Fisher L, Fukami N, Harrison ME, Ikenberry SO, Khan K, Krinsky ML, Maple J, Fanelli RD, Strohmeyer L. The role of endoscopy in the management of patients with peptic ulcer disease. Gastrointest Endosc. 2010 Apr;71(4):663-8.
7. Malfertheiner P, Megraud F, O'Morain CA, Gisbert JP, Kuipers EJ, Axon AT, Bazzoli F, Gasbarrini A, Atherton J, Graham DY, Hunt R, Moayyedi P, Rokkas T, Rugge M, Selgrad M, Suerbaum S, Sugano K, El-Omar EM., European Helicobacter and Microbiota Study Group and Consensus panel. Management of Helicobacter pylori infection-the Maastricht V/Florence Consensus Report. Gut. 2017 Jan;66(1):6-30.
8. Strand DS, Kim D, Peura DA. 25 Years of Proton Pump Inhibitors: A Comprehensive Review. Gut Liver. 2017 Jan 15;11(1):27-37.
9. Sachdeva AK, Zaren HA, Sigel B. Surgical treatment of peptic ulcer disease. Med Clin North Am. 1991 Jul;75(4):999-1012.
10. Chatila AT, Bilal M, Guturu P. Evaluation and management of acute pancreatitis. World J Clin Cases. 2019 May 06;7(9):1006-1020.