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Research Article

Investigation on Glycemic Imbalance Among Students Presenting with Symptoms of Gastritis in Maflekumen School of Health Sciences of Tiko-Cameroon

[1]Samgwa Osseiny, *[2]Esoh Rene Tanwieh, [3]Uani Ganyap Ismaella Sorel, [4]Fonyuy Marcel, [5]Solomon Gyampoh

About Article

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About Author

[1] Faculty of Medicine and Pharmaceutical Sciences, University of Douala, Cameroon

[2] Department of Medical and Biomedical Sciences, Faculty of Health Sciences, University of Bamenda, Cameroon

[3] Department of Medical Laboratory Sciences of Maflekumen Higher Institute of Health Sciences Tiko, Cameroon

[4] Department of Medical Laboratory Sciences of Maflekumen, Higher Institute of Health Sciences Tiko, Cameroon

[5] School of Pharmaceutical Sciences, Lovely Professional University, Jalandhar-Delhi G.T. Road, Phagwara, Punjab 144401, India

Contact @ Esoh Rene Tanwieh
esohrene999@gmail.com

ABSTRACT

Gastritis is an inflammation of the gastric mucosa. In 1728, Georg Ernest Stahl, a German physician first used the term "gastritis" to describe inflammation of the inner lining of the stomach. Inflammation refers to a physical condition in which a part of the body becomes irritated, reddened, swollen, and often painful. The objectives of this study were to investigate hyperglycemia, hypoglycemia, and euglycemia in Maflekumen students presenting with symptoms of gastritis. A laboratory based cross sectional study was carried out from December 2022 to March 2023. Fifty (50) students presenting with symptoms of gastritis were selected using a convenient sampling technique. A test strip was insert into the glucometer, and a small needle called lancet, provided with the test kit was used to prick the left side of the fingertip. Then, the edge of the strip was touch to drop of blood. And the blood sugar level of the participants displayed on the glucometer's screen after a few seconds. The findings of this study revealed that 16 (32%) of students presenting with gastritis symptoms were having high blood glucose concentration hyperglycemia, 22 (44%) of students were having hypoglycemia and 12 (24%) showed euglycemia. Conclusively, majority of students presenting with symptoms of gastritis were having low glucose level in their blood (hypoglycemia) which makes glycemic imbalance an important factor on the virulence of gastritis disease. A constant control of the glycemic level is recommended to avoid harmful and complication of the disorder.

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1. INTRODUCTION

1.1. Background

Gastritis is an inflammation of the gastric mucosa. In 1728, Georg Ernest Stahl, a German physician first used the term "gastritis" to describe inflammation of the inner lining of the stomach, the most common cause of gastritis being Helicobacter pylori (*H. pylori*) (Hellstrom, 2006).

H. pylori infect over 50% of the world's population and is a cause of chronic bacterial infections. Approximately 10% of infected individuals develop over clinical disease while 90% remain subclinical untreated. About 50% of the world population is infected by *H. pylori* and this rate of infection is higher in undeveloped countries as Cameroun, than in developed countries (de Brito *et al*., 2019).

In 1990, a new classification of gastritis called the Sydney system was presented to the world congress of gastroenterology in Sydney and was later published as six papers in the journal of gastroenterology and pathology (Sipponen, 2011). In 2005 gastritis staging using the OLGA (Operative link on gastritis assessment) staging system for reporting gastric histology was introduced (Stolle, 2001). Staging combines the atrophy score which is determined by biopsy mapping. Gastritis depending in the causes may be classified into acute gastritis, chronic gastritis, and atrophic gastritis (Rugge *et al*., 2008).

Helicobacter pylori infection occurs when *H. pylori* infect the stomach, transmitted through ingestion of contaminated food, water., Symptoms of *H. pylori* may include: an ache or burning pain in the stomach, nausea, loss of appetite, frequent burping, and bloating, unintentional weight loss (Yucel, 2014). *H. pylori* infection increases secretion of proinflammatory, cytokines, leading to changes in the structure of insulin receptors, disrupting the interaction between insulin and its receptors, and altered metabolism of glucose may generate chemical changes in the gastric mucosa that help to anchor *H. pylori* infraction (Kao *et al*., 2016).

1. Complications associated with *H. pylori* infection include / gastric ulcers, chronic gastritis and stomach cancer (Singhania, 2021). Common risk factors in the development of gastritis include: crowded and unsanitary living conditions, living without a reliable supply of clean water, living with someone who has an *H. pylori* infection (Zhu *et al*., 2014]

1.2. Rationale

Studies have estimated that approximately 20% of people infected during childhood will develop an ulcer, and that 2 to 3 % will be affected by cancer of the stomach. *H. pylori* infection according to studies increases secretion of proinflammatory cytokines, leading to changes in the structure of insulin receptors, disrupting the interaction between insulin and its receptors, and altered metabolism of glucose may generate chemical changes in the gastric mucosa. It is therefore important to conduct research to understand how gastritis exerts its harmful effect, and how blood sugar level affects the virulence of gastritis disease which will go a long way to reduce certain complications like stomach cancers associated with the infection.

1.3. Goal of Study

To educate and create awareness on the prevention of gastritis induced by *H. pylori* and associated risk factors that could result to glycemic imbalance.

1.4. Objectives

1.4.1. General objective

To evaluate glycaemia imbalance among students presenting with symptoms of gastritis.

1.4.2. Specific objectives

- To investigate hyperglycemia in Maflekumen students presenting with symptoms of gastritis
- To investigate hypoglycemia in Maflekumen students presenting with symptoms of gastritis
- To investigate euglycemia in Maflekumen students presenting with symptoms of gastritis

1.5. Operational Definition of terms

- Euglycemia: Normal concentration of glucose in blood (<70 mg/dL - >140 mg/dL).
- Hypoglycemia: Low concentration of glucose in blood (<70 mg/dL).
- Hyperglycemia: High concentration of glucose in blood (>140 mg/dL).

2. LITERATURE REVIEW

2.1. Anatomy of the stomach

The stomach is roughly J-shaped, although it sizes and position vary considerably, depending on the position of the body, the phase of respiration and whether it is full or empty. It also varies markedly with the build of the subject. In the asthenic individual it is elongated and, when full, may descend to the suprapubic region, while in the obese, plethoric subject it may be tucked away, in the lying position (Ellis, 2011).

The stomach has an anterior and posterior surface, a greater and lesser curvature and two orifices, the cardia, or, more accurately termed, the cardiac orifice, and the pylorus. The thick circular muscle of the pyloric sphincter is easily felt. However, in man there is no anatomical sphincter to be demonstrated at the cardia. The number of mechanisms responsible for the integrity of the cardiac is discussed in the article on the esophagus (Eker, 1951).

Along the lesser curvature of the stomach is a distinct notch, the incisura angularis, which is produced by the arrangement of the involuntary muscle fibers of the stomach wall (Latarjet & Wertheimer, 1921).

The fundus is the dome-like projection of the stomach above and to the left of the cardiac orifice. The body part of the stomach passes from the cardiac orifice to the incisura-it is this part of the organ that contains the parietal cells which secret HCL (Lamers, 1987). From the incisura to the pylorus is the pyloric antrum, which produces the hormone gastrin, responsible for the hormonal phase of gastric acid secretion. The pylorus is easily identified by palpation of the very distinct ring of sphincter crosses at this level (Sobala *et al*., 1991).

2.1.1. Physiology of the stomach

1. Gastric secretions: The cells of the gastric glands secrete about 2500 ml of gastric juice daily. This contains a variety of substances and gastric enzymes, whose role is to kill ingested bacteria, aid protein digestion, stimulate the flow of biliary and pancreatic juices and provide the necessary pH for pepsin to begin protein degradation (Talleey *et al.*, 1988).

2. Mucus secretion: The most abundant epithelial cells are mucus-secreting columnar cells, which cover the entire luminal surface and extend down into the glands as "mucous neck cells" (Tenca *et al.*, 2016).

3. Pepsinogen secretion: The chief cells secrete pepsinogens, contained in zymogen granules. These are the precursors of the pepsins (proteases) in gastric juice. Once secreted, pepsinogen 1 is activated by the presence of gastric acid into the active protease pepsin (Carabotti *et al.*, 2017).

4. Hormone secretion: The principal hormone secreted from the gastric epithelium is gastrin, a peptide that is important in control of acid secretion and gastric motility (Nomura *et al.*, 2014)

2.2. Gastritis

Gastritis is a medical condition where the lining of the stomach becomes inflamed. Inflammation refers to a physical condition in which a part of the body becomes irritated, reddened, swollen, and often painful. It may occur as a brief and sudden attack, as an ongoing condition, or as part of a medical illness., Inflammation of the whole stomach is called Pangastritis and an inflammation of a part of stomach is called Antral gastritis (Sierra, 2018).

2.2.1. Epidemiology of gastritis

1. Generally, most cases of gastritis are treatable once the etiology is determined. The exception to this is phlegmonous gastritis, which has a mortality rate of 65%, even with treatment. No sexual prediction exists, and it affects all age groups (Kao *et al.*, 2016). The incidence of *H. Pylori.* Infection increases with age. *H. pylori* infect over 50% of the world's population and is a cause of chronic bacterial infections (Singhania, 2021). Approximately 10% of infected individuals develop over clinical disease while 90% remain subclinical untreated. About 50% of the world population is infected by *H. pylori* and this rate of infection is higher in undeveloped countries as Cameroun, than in developed countries (Zhu *et al.*, 2014).

2.2.3. Classification of gastritis

Table 1. Topography, types and morphology of gastritis

Topography	Type	Morphology
-Antral	-Acute	-Active
-Fundal	-Chronic	-Atrophic
-Pangastritis	-Special forms: hypertrophic, granulomatous, lymphocytic, eosinophilic, reactive	-With intestinal metaplasia

1. Acute gastritis: Acute gastritis is a sudden inflammation or swelling in the lining of the stomach. It can cause severe and nagging pain (Sullivan, 2023). However the pain is temporary and usually lasts for short bursts at a time. It can be caused by injury, bacteria, viruses, stress, spicy food (Peyman *et al.*, 2003).

2. Chronic gastritis: Chronic gastritis is a chronic progressive stomach disease, pathological base of which are dystrophy, inflammation, disregeneration of gastric mucosa with atrophic as the outcome of these events. These conditions are accompanied by secretion, motoric and excretory function disturbances (National Library of Medicine, 2022).

2.3. Etiology of gastritis

Gastritis has a number of causes, including the following:

- Bacterial infections: *H. pylori* (mostly), staphylococcus, alpha hemolytic streptococcus
- Non-steroid anti-inflammatory drugs (NSAID)
- Autoimmune: Atrophic gastritis with Biemer anemia
- Bile reflux disease
- Stress
- Alcohol addiction
- Cocaine
- Radiation and chemotherapy

2.3.1. Clinical manifestation of gastritis

Not everyone with gastritis will experience symptoms identically. Some persons will have:

- Pain or discomfort in the upper abdomen,
- nausea or vomiting,
- feeling full too soon during a meal,
- loss of appetite, weight loss, bloating,
- severe chest pain,
- feeling faint or short of breath

2.4. Glycaemia

Glycaemia, also known as blood sugar level refers to the glucose concentration in the blood. Blood glucose is one of the most important blood parameters to measure (in mg/dL) (CDC, 2022). The blood 's concentration of glucose varies during the day and night. But throughout the day, our bodies work to keep the amount of blood glucose constant. A healthy body has an average blood sugar level of 90 to 100 mg/dL (Mathew *et al.*, 2023) However, there are instances when these blood sugar levels can change as a result of other things, like illnesses. An abnormality in blood sugar stability can include: Hypoglycemia (low blood sugar) or Hyperglycemia (high blood sugar).

2.4.1. Effects of hyperglycemia on gastritis

Hyperglycemia increases the risk of gastric cancer in Pylori infected patients. High glucose could increase endothelial permeability and cancer-assisted signaling. Higher glucose could maintain *H. pylori* growth (George, 1968).

1. HbA1c is the major fraction of GHB and results from non-enzymatic glycosylation. HBA1c reflects the integrated blood glucose levels (Carabotti *et al.*, 2017). Hyperglycemia reduces gastrin stimulated gastric acid secretion in humans. Hyperglycemia has a profound effect on gastric motility it reduced the stomach motility (Carabotti *et al.*, 2017). Gastric emptying is highly sensitive to acute changes in the blood

glucose concentration (Latarjet, 1921). Acute hyperglycemia (i.e., blood glucose level 15 mmol/l (270 mg/dl)) substantially slows gastric emptying (Rugge, 2008). indeed, even changes within the normal range of postprandial glycaemia have marked effects on the rate of gastric emptying, with emptying being slower when blood glucose is clamped at 8mmol/L (144 mg/dL) compared with emptying rates at 4mmol/L (72 mg/dL) (Sun & Shay, 1960).

Gastric acid output was measured with a recovery marker (phenol red) under basal conditions and after repeated intragastric instillation. During normoglycemia. Premeal gastric acid output was significantly (P<0.05) reduced during hyperglycemia compared with during normoglycemia. (2.6+/-1.0 vs. 5.8+/-1.8 mmol/h). Intragastric meal stimulated incremental acid output during hyperglycemia was significantly (P < 0.05) reduced compared with that during normoglycemia (4.9+/-1.3 vs. 6.6+/- 1.6 micrograms (-1).120 min-1). During hyperglycemia, recovery rates of gastric contents were significantly (P < 0.005) increased compared with during normoglycemia, both before (81+/- 4 vs. 71+/- 6 %) and after (72+/- 4 vs. 57+/- 4%) meal ingestion, pointing to delayed gastric emptying of liquids during hyperglycemia (Lam *et al.*, 1998).

Older work in man with meals of carbohydrates in water has indicated that such meals slow gastric emptying in proportion to their osmolarities (Meeroff *et al.*, 1973). Nevertheless, different efficacies per millions mole. One possibility which would explain such discrepancies among carbohydrates is that hyperglycemia induced by carbohydrate absorption itself contributes to the slowing of gastric emptying. To test this possibility, normal subjects were made acutely hyperglycemic with intravenous loads of glucose during the ingestion of various liquid test meals, and rates of gastric emptying euglycemia conditions(George, 1968; Malawer & Powell, 1967)

Induced hyperglycemia significantly slowed the rate of emptying of meals containing fat+ protein, or protein, but did not significantly alter emptying of meals containing only NaCl. It is concluded that hyperglycemia does exert some effect on gastric emptying, but that these effects of hyperglycemia are variably expressed (George, 1968).

Hyperglycemia may augment gastric colonization by *H. pylori* and aggravate the resulting gastritis and mucosal atrophy (Fraser *et al.*, 1991).

H. pylori adhesion and Cag A increased to further facilitate the enhancement of cell-associated Cag A in higher glucose conditions (Marathe *et al.*, 2013).

2.4.2. Effects of hypoglycemia on gastritis

Low blood sugar levels affect the signaling of the vagus nerve, which normally stimulates the stomach to empty. Gastro paresis causes problems with the digestive system because it can cause food to spend too much time in the stomach before entering the small intestine manifested by nausea, vomiting and stomach pain (Sullivan, 2023; Sun & Shay, 1960). Helicobacter pylori induced gastritis may contribute to occurrence of postprandial symptomatic hypoglycemia.

In the condition so far found to increase gastric hunger contractions there is either a decrease in tissues glycogen, or inability of the tissue to burn sugar. Furthermore, one of the symptoms accompanying insulin hypoglycemia in man is increased hunger (Carlson, 1914).

The effect of alterations of blood glucose levels on gastric acid in man. The effect of procedures which alter blood glucose, i.e., infusion of 0.2 units/kg body wt/hr insulin and/or 0.66g/kg body wt/hr glucose, on gastric acid secretion(Powell & Hirschowitz, 1964). Each subject underwent experimental procedures. Acid output was measured continuously be means of intragastric titration (Saltiel, 2021). Low glucose levels were associated with high rates of acid secretion. These results reveal a determining influence of blood glucose levels on acid secretion.

Results of the study show three components of the mechanism of action of insulin hypoglycemia on gastric secretion, an initial inhibitory effect on basal gastric secretion, and a stimulating effect mediated through the adrenal glands manifested in the late phase of secretion. However, the magnitude of this late of gastric secretion was considerably less pronounced after vagotomy. A significant hypoglycemia of 40 mg% or less was necessary to produce the late phase of gastric secretion. The adrenal phase can contribute to the potential for ulcer occurrence or reactivation, not so much through the increase in secretion it alone might induce (Sun & Shay, 1960).

It is now appreciated that gastric emptying is a major determinant of the glycemic response to carbohydrate-containing meals in both health and diabetes, and that acute hypoglycemia accelerates gastric emptying substantially. However, the potential relevance of gastric emptying to the predisposition to, and counter-regulation of, hypoglycemia has received little attention, (Serrano-Castro, 2022). In insulin-treated patients, the rate of gastric emptying induced by hypoglycemia probably represents an important counter-regulatory response to increase the rate of carbohydrate absorption (George, 1968).

2.5. Risk factors associated with gastritis

The risk of developing gastritis is at its highest when:

- A person falls ill with a viral, parasitic or bacterial infection. A vulnerability to Helicobacter pylori can sometimes be inherited or caused by certain lifestyle choices exposing you to the bacteria or that which makes you more vulnerable. Such choices include smoking and allowing high levels of continuous stress in your life. The stomach lining things naturally as a person ages. Older adults are also typically more prone to Helicobacter pylori or autoimmune disorders than younger people are. Person indulges in extreme alcohol irritates the stomach lining and thus causes disruptions when in contact with stomach juices.

- Person regularly uses non-steroidal anti-inflammatory medications such as aspirin and ibuprofen. Someone who has autoimmune disorder or digestive disorder. A person with other medical conditions such as HIV/AIDS and Crohn's disease [Cogn Med Sci.1958]

3. METHODOLOGY

3.1. Study Area and Setting

The study was carried out in Maflekumen School of Health Sciences. Maflekumen is found in TIKO subdivision under FAKO division in the Southwest Region of Cameroon. Many students are affected by gastritis due to the consumption of contaminated food and water around the school campus and community.

3.2. Study Design and Duration

The study was a cross-sectional study for duration of four (4) months from December 2022 to March 2023.

3.3. Study Population and Sampling

The study was carried out on 50 students of Maflekumen School of health sciences Tiko presenting with symptoms of gastritis. A convenient sampling technique was used to achieve the sample.

3.4. Inclusion / Exclusion Criteria

3.4.1. Inclusion criteria

Students suffering from gastritis.

3.4.2. Exclusion Criteria

Students not belonging to Maflekumen and students on treatment of gastritis

3.5. Ethical Consideration

An introductive letter to carry out research was obtained from the CEO of Maflekumen School of Health Sciences Tiko, and, an administrative authorization to collect research data from the DMO of Tiko District.

3.6 Data Collection and Technique

3.6.1. Data Collection

A fingertip capillary blood was collected from participants and analyzed using a glucometer. The test is based on the measurement of electrical current generated by the reaction of glucose with the reagent of the strip. The meter measures the current and displays the corresponding blood glucose level. The strength of the current produced by the reaction depends on the amount of glucose in the blood sample. Requirements: gloves, cotton, 70% alcohol, lancet, blood sugar level meter (Glucometer).

3.6.2. Blood sugar level measurement Technique

1. Wash my hands with soap and warm water and before wearing gloves.
2. Removed a test strip from its vial. With clean, dry hands, you may touch the test strip anywhere on its surface. Do not bend, cut or modify the test strips in any way. Remove the test strip from the vial and use it instantly.
3. Insert the test strip into the meter's test port and the meter is turned on.
4. When the blood drop symbol flashes on the screen; you may select the appropriate measurement mode.
5. Disinfected the fingertip with 70% alcohol and by the use of a lancet, pricked the fingertip.
6. Apply blood to the absorbent hole of the test strip.
7. As soon as enough blood has filled the confirmation window of the test strip heard a beep letting you know the test has begun. A countdown of 5 seconds starts.
8. blood glucose level appears on the display.
9. Eject the used test strip and remove the lancet. To eject the test strip, point the strip at a disposal container for sharp objects.
10. Wash hands thoroughly with soap and water.

3.7. Data Analysis

Calculations were done on Microsoft excel version 2010, and represented by tables, and bar charts.

4. RESULTS AND DISCUSSION

4.1. General characteristics of Maflekumen students presenting with symptoms of gastritis

The study was carried out on 50 students of Maflekumen School of health sciences Tiko presenting with symptoms of gastritis made of 16 (32%) males and 34 (68%) females.

The study was carried out on 50 students of Maflekumen School of health sciences Tiko presenting with symptoms of gastritis made of 16 (32%) males and 34 (68%) females.

Table 2. General characteristics of students

Gender	Age range	Students with Hyperglycemia (>140 mg/dL)	Students with Hypoglycemia(<70 mg/dL)	Students with Euglycemia (>70 mg/dL <140 mg/dL)	Total Of Participants (Male + female)
Male	22-30 years	05 (10%)	07 (14%)	04 (08%)	16 (32%)
Female	20-28 years	11 (22%)	15 (30%)	08 (16%)	34 (68%)
General Total		16 (32%)	22 (44%)	12 (24%)	50 (100%)

4.2. Hyperglycemia in Maflekumen students presenting with symptoms of gastritis

The current study on 50 students of Maflekumen School of health sciences Tiko presenting with symptoms of gastritis revealed that 16 (32%) of the students had high blood sugar level (>140 mg/dL).

Out of a total of 50 students of Maflekumen School of health sciences Tiko enrolled presenting with symptoms of gastritis22 (44%) of student participants with gastritis had low blood sugar level (<70 mg/dL).

4.3. Euglycemia in Maflekumen students presenting with symptoms of gastritis

Out of a total of the 50 students of Maflekumen presenting with symptoms of gastritis, 12 (24%) of the participants presented with normal blood sugar level (<140 mg/dL).

4.4. Discussion

Gastritis is an inflammation of the gastric mucosa. In 1728, Georg Ernest Stahl, a German physician first used the term "gastritis" to describe inflammation of the inner lining of the stomach, the most common cause of gastritis being Helicobacter

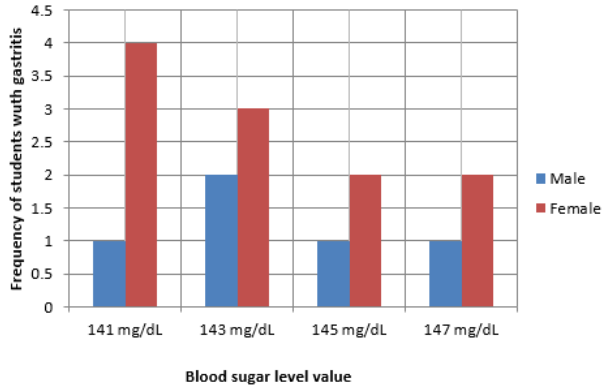

Figure 1. Hyperglycemia in gastritis individuals

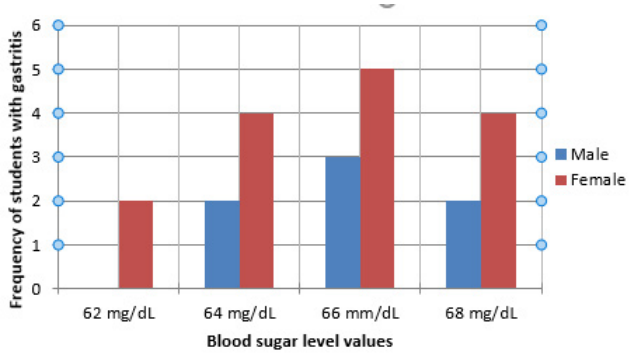

Figure 2. Hypoglycemia in gastritis individuals

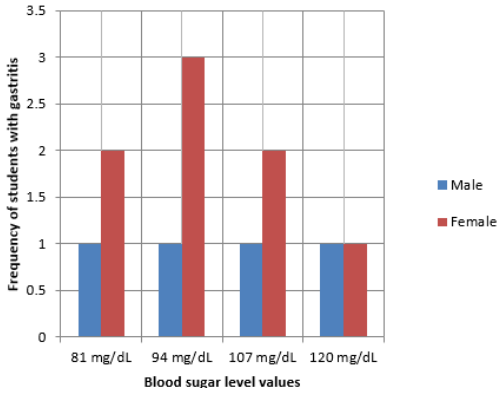

Figure 3. Euglycemia in gastritis individuals

pylori (*H. pylori*) (Hellström, 2006). *H. pylori* infect over 50% of the world's population and is a cause of chronic bacterial infections. Approximately 10% of infected individuals develop over clinical disease while 90% remain subclinical untreated. The objectives of this study were to investigate hyperglycemia, hypoglycemia, and euglycemia in Maflekumen students presenting with symptoms of gastritis.

4.4.1. Hyperglycemia in Maflekumen students presenting with symptoms of gastritis

According to the findings of our current study, out of a total fifty (50) participants presenting with symptoms of gastritis, many of the students (32%) were having a significantly high blood sugar level ranging between [141 mg/dL – 147 mg/dL]. This result is similar to another study carried out by Lam *et al.* (1998) in May whereby gastric acid output was measured with a recovery marker (phenol red) under basal conditions and after repeated intragastric instillation during normoglycemia. Premeal gastric acid output was significantly ($P<0.05$) reduced during hyperglycemia compared with during normoglycemia. (2.6+/-1.0 vs. 5.8+/-1.8 mmol/h). Intragastric meal stimulated incremental acid output during hyperglycemia was significantly ($P < 0.05$) reduced compared with that during normoglycemia (4.9+/-1.3 vs. 6.6+/- 1.6 micrograms (-1).120 min-1). During hyperglycemia, recovery rates of gastric contents were significantly ($P < 0.005$) increased compared with during normoglycemia, both before (81+/- 4 vs. 71+/- 6 %) and after (72+/- 4 vs. 57+/- 4%) meal ingestion, pointing to delayed gastric emptying of liquids during hyperglycemia (Lam *et al.*, 1997). The similarity could be due to the fact that their rate of gastric emptying is slowed down because of the high concentration of glucose in their blood leading to some symptoms of gastritis like bloating.

4.4.2. Hypoglycemia in Maflekumen students presenting with symptoms of gastritis

According to the second specific objective, a significantly very high number of students 22 (44%) presenting with symptoms of gastritis were having low concentration of glucose in their blood, the range comprised between 62 mg/dL – 68 mg/dL. This result was also similar to that showed that low glucose levels were associated with high rates of acid secretion. These results reveal a determining influence of blood glucose levels on acid secretion (Eker, 1951). Also, Sun & Shay (1960) indicated three components of the mechanism of action of insulin hypoglycemia on gastric secretion, an initial inhibitory effect on basal gastric secretion, a stimulating effect mediated through the adrenal glands manifested in the late phase of secretion. However, the magnitude of gastric secretion was considerably less pronounced after vagotomy. A significant hypoglycemia of 40 mg% or less was necessary to produce the late phase of gastric secretion. The adrenal phase can contribute to the potential for ulcer occurrence or reactivation, not so much through the increase in secretion it alone might induce (Sun & Shay, 1960). This could be due to the fact the hypoglycemia is the main stimulus of gastric acid secretion causing gastritis symptoms like chest pain.

4.4.3. Euglycemia in Maflekumen students presenting with symptoms of gastritis

According to our current study, majority of students 12 (24%) presenting with symptoms of gastritis were having normal blood sugar level ranging between (81 mg/dL-120 mg/dL). This result was similar the study carried out by which was by a glucose-insulin clamp technique blood glucose levels were kept constant during the studies at 5.0mmol/liter (euglycemia clamp), or 7.0 mmol/liter on three different days. Glucose and insulin were not infused during one control day study. The present observation indicates that the inhibitory effect of glucagon is lost when blood glucose is below a certain limit, suggesting that blood glucose may have a modulating effect on gastric acid secretion.

6. CONCLUSIONS

Conclusively, based on our investigation of glycemic imbalance

among fifty (50) students presenting with gastritis symptoms attending Maflekumen School of health sciences Tiko, majority of the participants 22 (44%) presented with hypoglycemia 62 mg/dL – 68 mg/dL. Furthermore, a moderately high number 16 (32%) of the student participants were hyperglycemic 141 mg/dL – 147 mg/dL. Finally, a few participants 12 (24%) presented with euglycemia.

RECOMMENDATIONS

Based on this study, the following are recommended:

1. Perform regular control of glycemic level
2. Avoid starvations
3. Food and water hygiene
4. Avoid eating smaller meals throughout the day
5. Avoid fatty, fried, spicy or acidic foods
6. Cut back caffeine
7. Not lying down for 2 to 3 hours after a meal
8. Avoid taking NSAIDs
9. Reducing alcohol consumption

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