

## Determination the Level of Complement Protein C3 and C4 in Gastric Ulcers Patients in Baqubah City, Iraq

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### Abstract

**Background:** Gastric ulcer is common and serious diseases caused by imbalances between naturally occurring defensive factors such as mucus, prostaglandins and aggressive factors such as hydrochloric acid, usually found in gastrointestinal juices

**Objective:** To evaluated the level of complement protein C3 and C4 for infected patients with gastric ulcer in Baqubah city, Iraq.

**Patients and Methods:** In this case- control study (57) blood samples were collected from patients with gastric ulcer and (32) blood samples from healthy individuals has been accredited as a control group and the study has continued during the period between October 2016 and April 2017.

**Results:** The results showed that there was a decrease in the level of C3 in patients with gastric ulcers by  $(25.168 \pm 182.173)$  mg/dl compared to the control group and  $(501.565 \pm 481.418)$  mg/dl and found that an increase in the level of C4 in patients with gastric ulcer was  $(34.978 \pm 91.252)$  mg/dl compared to control group and  $(7.493 \pm 17.403)$  mg/dl in (p value < 0.001).

**Conclusion:** We conclude from this that the complemental protein plays an important role in the localized response to gastric ulcer and gastrointestinal diseases. The presence of neutrophil cells leads to an increase in the production of the C4 complement in patients and that IL-10 has a role in regulating the complemental product in general .

**Key words:** Complement Protein , C3 and C4, Gastric Ulcer.

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### Introduction

Gastric ulcers are inflammation of the lining of the stomach due to the secretion of acid and pepsin in the gastric juices. This causes the health and functional disorders of the stomach and depends on the disease on the type of pathogen and its effectiveness, in addition to the strength of the cellular immune response and host [1]. Among the most prevalent diseases in developing countries than in developed countries and at all ages, 35% of adults in

the United States of America and 15% of children were diagnosed at the age of 2-8 years(2). There are many factors that contribute to the development and development of Gastric ulcers[3], The most important and most common bacteria Helicobacter Pylori which is a major cause of gastric ulcer [4]. The treatment of non-steroidal anti-inflammatory drugs and chemicals harmful to gastric mucosa, yellow components, Stress and pancreatic

juice (5,6). Symptoms of stomach ulcers appear abdominal pain can vary from person to person, some people have no pain, other symptoms include feeling full and drinking plenty of fluids as usual, feeling hungry often after 1-3 hours after meal, mild nausea may disappear With vomiting, pain or discomfort at the top of the abdomen can awaken the patient at night (7,8).As well as the role of immunological factors that urge gastritis to an inflammatory response in the tissues of the mucous infectious and linked to the immune processes consistent and the liberalization of these factors play a major role in increasing the risk of stomach disease(9). These factors are complement proteins that play a role in stimulating the immune response against many diseases, including gastric ulcers, which mediate H.pylori bacteria through the alternative path. The most important of these proteins C3 and C4[10].

## Patients and Methods

### The Study samples

A total of 57 blood samples were collected from patients suffering from gastric ulcers based on the clinical diagnosis by the medical staff at Baqubah General Education Hospital in Diyala Governorate and 32 blood samples from healthy people and their adoption as a control group. The study lasted from the restricted period Between November and April 2017.

**Table (1):** Comparison of complementary proteins C4, C3 with the two study groups.

mg/dl		Study group		P value
		Control	Patients	
C3	Mean	481.418	182.173	0.000
	SD	501.565	25.168	
C4	Mean	17.403	91.252	0.000
	SD	7.493	34.978	

\*There is a significant statistical difference  $p < 0.001$

## Measurement of C3 & C4 complement proteins using a technique single radial inamuuodiffusion plate

The level of C3 and C4 supplemental components was estimated using SIR technique, in which the complement component of the study sample was correlated with the nonspecific antibodies found in the agarose gel, where the immune complex that forms a precipitin ring is formed in the shape of a ring around the hole and the diameter The loop is proportional to the concentration of the decomposed protein and this occurs within 72 hours of incubation[11].

### Statistical Analysis

We analyzed the data of all tests statistically using the calculator and using the Statistical Package of Social Science (SPSS). We rely on the T-Test at a probability level (0.01) to determine the significance of the differences between the transaction rates[12]

### Results

The results of the present study showed a decrease in the level of C3 in patients with gastric ulcers ( $182.173 \pm 25.168$ ) Mg/dL compared with the control group ( $481.418 \pm 501.565$ ) mg/dl and a significant statistical significance difference ( $0.001 < p$ ).

In the statistical comparison between the studied groups, the statistical analysis showed a significant difference in gastric ulcer patients compared to control group.

## Discussion

These results were in line with the findings of the researcher Berstad and others (13), which indicated that there is a C3 tonic in small amounts on the surface of epithelial cells in the stomach where he found that C3 exists in small quantities in the body and gastric cavity in patients with gastritis caused by H.pylori bacteria or Without them, and this may explain why it is low in the incidence of gastric ulcer patients covered. (14) Al-Any showed that C3 did not rise in patients with gastric ulcer compared to healthy patients. Another study also showed a decrease in C3 in a study of the exacerbation of H. pylori infections (15).

The results of the current study showed that the level of serum C4 complement in the serum of gastric ulcer patients increased by  $(34.978 \pm 91.252)$  compared to the control group  $(7.893 \pm 17.403)$ . This increase was obtained with high significance ( $P < 0.001$ ). The results of this study agree with the findings of Ismail and his group (16) that the infection of H.pylori bacteria in the case of chronic gastritis urges to increase the level of C4 in the blood of patients, which lead to the activation of the complement system through the classical path, and the presence of cells of neutrophil the stimulant will increase C4 complemental output in patients compared with healthy patients, and suggests that the IL-10 mediator has an effective role in regulating overall complementarity. Another study indicates that the high level of complement C4 in patients with gastrointestinal diseases, chronic gastritis and chronic gastritis, especially as the serological level of the complement component C3, and stressed the importance of complement system in diseases of chronic gastritis This is evidenced by the important role played by the complement system in the localized immune response to chronic gastritis and the presence of H. pylori and intestinal diseases (17). But this study does not agree with the

study of the researcher Amer et al., that indicate a decrease in the level of C4 in patients infected with H.Pylori bacteria causing gastric ulcers (18). We conclude from this that there is a decrease in the level of c3 and a rise in the level of c4 in the serum of patients with gastric ulcers due to the important role that performs the system of mutant in the body, which performs many functions, namely chemical attraction and control of inflammatory reactions and the elimination of the complexes of immune and cellular activation and anti-microbial defenses.

## References

- [1] Al-Howiriny T. Alsheikh A. Alqasoumi S.; Al-Yahya M.; ElTahir K. and Rafatullah S. Gastric antiulcer, antisecretory and cytoprotective properties of celery (*Apium graveolens*) in rats. *J Ethnopharmacol* . 2010 ;48(7):786-93. Doi:10.3109/13880200903280026. PMID:20645778.
- [2] Bauer B. and Meyer T F. The Human Gastric Pathogen *Helicobacter pylori* and Its Association with Gastric Cancer and Ulcer Disease. Hindawi Publishing Corporation *Ulcers*. 2011 Volume . Article ID 340157, 23 pages doi:10.1155/2011/340157.
- [3] Schubert ML. and Peura DA. Control of gastric acid secretion in health and disease . *J Gastroenterology*. 2008; 134(7):1842-1860
- [4] Majumdar D. Bebb J. and Atherton J. *Helicobacter pylori* infection and peptic ulcers. *Medicine*. 2011; 39, p.p. 154-161.
- [5] Yeomans ND. The ulcer sleuths: The search for the cause of peptic ulcers. *J Gastroenterology and Hepatology*. 2011; 26 Suppl 1: 35–41. doi:10.1111/j.1440-1746.2010.06537. x. PMID 21199512.
- [6] Fink G. Stress controversies: post-traumatic stress disorder, hippocampal volume, gastroduodenal ulceration. *J neuroendocrinology*. 2011 ; 23 (2): 107–17.

doi:10.1111/j.1365-2826.2010.02089.x.

PMID 20973838.

[7] Chan FKL. and Lau JYW. Peptic ulcer disease. In: Feldman M, Friedman LS, Brandt LJ, eds. *Sleisenger and Fordtran's Gastrointestinal and Liver Disease*. 2016; 10th ed. Philadelphia, PA: Elsevier Saunders; chapter 53.

[8] Morgan DR. and Crowe SE. *Helicobacter pylori infection*. In: Feldman M, Friedman LS, Brandt LJ, eds. *Sleisenger and Fordtran's Gastrointestinal and Liver Disease*. 2016 ; 10th ed. Philadelphia, PA: Elsevier Saunders;chapter 51.

[9] Mahdi BM. Role of immunity in gastric ulcer. *J. of gastroenterology and hepatology*. 2013; 2(10):803-806.

[10] Jean L. Bolongia M D. Joseph L. Jorizzo and Julie V. *British Library Cataloguing in Publication Data Dermatology*, 3rd (Ed). *Ebo Der*. 2012; 1:1-2561.

[11] Recasens M. Lopez-Bermejo A. and Ricart W. An inflammation score is better associated with basal than stimulated surrogate indexes of insulin resistance. *J Clin Endocrinol Metab*. 2005 ; 90 112-116.

[12] Nisi AD. *Statistical analysis in medical research*, 2nd (Ed). 2004; 22: 21-30 .

[13] Berstad AE. Brandtzaeg P. Stave R. and Halstensen TS. Epithelium related deposition of activated complement in *Helicobacter pylori* associated gastritis. *J Gut*. 1997; 40: 196-20.

[14] Al-Any EA. The Role of Interleukin 8 Associated with Peptic Ulcer Disease Induced by *Helicobacter pylori*. M.Sc. thesis, College of Science, Al-Mustansiriyah University, Iraq 2005.

[15] Keran JI. Liyun A. Fukun W. Lanchun S. Xiangyang R. Xianling W. Zhanguo H. and Jing C. Aggravation of *Helicobacter pylori* stomach infections in stressed military recruits. *J of International Medical Research*. 2016; 44(2) 367-376. DOI: 10.1177/0300060515593768.

[16] Ismail HF. Zhang J. Lynch RG.; Wang Y. and Berg DJ. Role for complement in development of *Helicobacter*-induced gastritis in interleukin-10 deficient mice. *J Infection and Immunity*. 2003; 71(12): 7140-7148.

[17] Copeland BH. Aramide OO. Wehbe SA. Fitzgerald SM and Krishnaswamy G. Eosinophilia in a patient with cyclical vomiting : a case report. *J Clin Mol Allergy*. 2004 ; 2:(7).

[18] Amer NA. Janan G. Hassan L. Ihsan E. Al-Saimary A. Current Concepts in the Immunological Diagnosis of *H. pylori* in Basrah Pediatric Oncology Unit. *Donnish J of Microbiology and Biotechnology Research*. 2014; 1(2) pp. 018-022.