Journal of Basrah Researches (Sciences) **49**(1), 23 (2023)
DOI: https://doi.org/10.56714/bjrs.49.1.3

Evaluation of IL-8 and IL-10 Levels in men with Irritable Bowel Syndrome

Suad M. Salman, Heba T. Yser*

Department of Biology, College of Education for Pure Sciences, University of Basrah, Basrah, Iraq.

ARTICLE INFO

Received 18 November 2022
Accepted 11 March 2023
Published 30 June 2023

Keywords:

Interleukin 8, Interleukin 10, IBS, Human.

Citation: S.M. Salman1, H.T. Yser, J. Basrah Res. (Sci.) **49**(1), 23 (2023).
DOI:https://doi.org/10.56714/bjrs.49.1.3

ABSTRACT

Irritable Bowel Syndrome (IBS) is a common disorder of the large intestine. This study was designed to Estimation of interleukin 8 and 10 in human suffering from irritable bowel syndrome and the relationship with age and period of disease. A total of 88 men participated in this study, including 57 men as patient, and 31 as healthy, who were collected from Al-Fayhaa Hospital in Basrah. The patients was classified according to age into (30-40), (41-50) and (51-60), and according to the disease period into (1-5) and (6-10) years. The healthy were aged (30-60 years).IL-8 and IL-10 were measured using ELISA technique and The data statistical analysis software (One-way Anova) and Mann-Watny test. The results showed a significant increase of interleukin-8 (IL-8) for patients at P≤0.001, while there was a significant decrease in the level of interleukin-10 (IL-10) at P≤0.001. Regarding the age groups, the results did not show variation of IL-8 levels .while, the level of IL-10 in the (41-50 years) was significantly lower. As for the relationship between the duration of the disease there is no significant differences. The study concluded that inflammations play a role in inducing irritable bowel syndrome and the age group (41-50) was more affected.

1. Introduction

Many people suffer from Functional gastrointestinal disorders (FGIDs), which are heterogeneous groups of chronic disease symptoms such as irritable bowel, and there is no specific standard for their diagnosis, nor a specific treatment is agreed-upon for irritable bowel treatment. However, developments in science and modern technology have contributed to understanding the complex relationship between the gut, the immune system and the nervous system together, which has led to many treatment options to relieve symptoms [1]. The Functional gastrointestinal disorders (FGIDs) can affect any part of the digestive system (GI) including the esophagus, stomach, and intestines. These disorders represent a huge economic and social burden; and they are fundamentally functional, not structural or biochemical abnormalities, nor are they considered psychiatric disorders, although stress and psychological difficulties can worsen the FGIDs symptoms [2,3] The part of the digestive canal most susceptible to infection is the colon, with a

***Corresponding author email :** heba.yser@uobasrah.edu.iq

ISSN: 1817-2695 (Print); 2411-524X (Online)
Online at: https://jou.jobrs.edu.iq

global prevalence estimated at 20% [4,5]. Irritable bowel syndrome is defined as a dysfunction of the colon, which leads to the appearance of symptoms in the digestive system, such as indigestion, bloating, constipation, and cramps that disappear after defecation; in addition to a feeling that the defecation is not completed after going to the bathroom, and sounds that the person close to the patient may hear [6,7]. There are many causes and factors that lead to irritable bowel syndrome. Among these factors that affect and are affected by colon disorders are inflammatory interleukins (a group of proteins belonging to a low molecular weight family with chemical activity to direct leukocytes [8], which are divided into several subfamilies that depend on the position of one or two cysteine residues involved in the formation of the molecular disulfide bond. Members of the chemokine family, or C-X-C, have a pair of cysteine residues separated by a single amino acid including interleukin-8 (IL-8). The interleukin-8 (IL-8) is considered as a protein containing 99 amino acid residues with the NH2 amino acid sequence differences among the types of cells from which it is released but the initial form of IL-8 contains 77 amino acids [9]. It is also considered as a chemical agent that attracts neutrophils, basophils, and T cells during the inflammatory process; not just a chemical agent for monocytes; as IL-8 is involved in the activation of neutrophils and is released from several types of cells in response to inflammations such as monocytes, macrophages, neutrophils, cells of the intestine, kidneys, placenta, and bone marrow. It is a member of the beta-thromboglobulin superfamily and is structurally related to the platelet factor. Endothelial cells store interleukin-8 in their storage vesicles (Weibel–Palade body) [10]. IL-8 is involved in mitosis, inhibition of angiogenesis, inflammation, chemotaxis, neutrophil lysis, leukocyte activation, and calcium homeostasis [11].

The study of Pandey and Rizvi (2009) [12], which included 38 patients with colon disease (32 females, 6 males; age range 18-70 years) and 22 healthy individuals (17 females, 5 males, age range 24-42 years) - where the diagnosis of Irritable bowel syndrome was done according to the Rome criteria – showed elevated proinflammatory cytokines such as IL-6 and IL-8.

The study of Zhen, et al., (2015) [13] included 42 patients and 20 healthy men, and aimed to explore the relationship between interleukin (IL-8) and abdominal symptoms and psychological factors in patients with diarrhea associated with irritable bowel syndrome, The results showed that the level of IL-8 was higher in the patients group compared to the healthy group. As for non-inflammatory interleukins, they also affect and are affected by irritable bowel syndrome; which are basically a group of cytokines (proteins/molecules) first seen in white blood cells that stimulate the body's immune system to resist congestion and disease. Interleukins are formed in a wide range of cells in the body, including white blood cells, which expel or destroy bacteria and other harmful substances that enter the body [14]. The immune system has developed anti-inflammatory mechanisms that limit the production of pro-inflammatory molecules to reduce tissue damage and maintain or restore tissues homeostasis. Anti-inflammatory interleukins play a critical and necessary role in the prevention of inflammatory diseases, autoimmune diseases, non-immune effect or types, epithelial cells and keratinocytes [15]. Interleukin IL-10 is a multidirectional cytokine protein that inhibits the synthesis of a number of cytokines such as interleukin-1 and interleukin-2. It is also known for its strong anti-inflammatory and immunomodulatory effects. IL-10 was originally identified as a product of T helper 2 cells [16]. However, it is actually produced by many immune cells derived from the spinal cord and lymphatics that participate in both natural immunity and adaptive immunity [17].

The study of Max Schmulson, *et al*., (2013)[18] included 137 volunteers, including 92 healthy and 45 patients with irritable bowel syndrome, who were divided into three subgroups: IBS-D, IBS-C, IBS-M, and there were no statistically significant differences in Age, gender, and body mass index (BMI). The study showed that the IL-10 level was low in patients compared to the healthy volunteers, but when comparing the subgroups, the IL-10 level was higher in IBS-C than in both IBS-M and IBS-D.

The study of Ellen Johanne Vara, *et al*. (2018)[19] which included 38 patients with Irritable Bowel Syndrome (32 females, 6 males, with the age range of 18-70 years) and 22 individuals representing the healthy group (17 females, 5 males, with the age range of 24 -42 years), showed that IL-10 level in serum was significantly lower in patients with irritable bowel syndrome

compared to the healthy group; and this cytokine imbalance may act as a possible causative factor for Irritable Bowel Syndrome.

2.Materials and working methods

2.1.Healthy Samples

The current study used 31 serum samples of healthy volunteers with age range (30-60 years), after confirmation from the specialist doctor that they do not complain from irritable bowel syndrome.

2.2.Patient Samples

A total of 57 serum samples were collected from patients of colon disorders with age range (30-60 years), classified according to disease time period into two categories: (1-5 years) and (5-10 years). The samples were collected from Al-Fayhaa Hospital and the Center of gastrointestinal tract and colon Diseases and surgery.

2.3. Serum preparation

Blood samples were taken from each volunteer when visiting the Center of Gastrointestinal Tract and Liver Diseases and Surgery and Al-Fayhaa Hospital which are run by Basrah Health Directorate. The specialist doctor examined each of the volunteers and sent him to the Laboratory Unit, where the laboratory technician drew approximately (5 ml) of venous antecubital blood. The next step, the blood was placed in a special Gel Tube and left for a period ranging between 10 and 15 minutes. Then, the samples were centrifuged at a rate of 4000 revolutions per minute for a period of 15 minutes to obtain the serum. The resulted serum was distributed among small Eppendorf tubes. then the samples were kept at (-20) ºC in deep freezing until the tests were conducted.

2.4. Estimation of the concentration of interleukins

A special kit manufactured by Elabscience Company were used with serial number (Catalog No: E-EL-H6008) for interleukin 8 (IL-8), and serial number (Catalog No: E-EL-H6154) for interleukin 10 (IL-10), through the Linked Enzyme Immunoassay (ELISA) technique.

3.Statistical Analysis

Statistical analysis of the data was performed using analysis of variance (ANOVA) for least significant difference in LSD at $P \leqslant 0.05$ and $P \leqslant 0.01$ The standard error was calculated using SPSS version 23.

4. Results:

4.1. Assessment of IL-8 and IL-10 levels in both the Patient and Healthy Groups

The results of the current study (detailed in Table 1) showed a significant difference in the IL-8 levels at a probability of ($P \leqslant 0.001$) between the patient group and the healthy group, as the level increased significantly in the patient group (24.473±2.099) compared to the healthy group (4.792±0.417).

As for the IL-10, the results of the current study (detailed in Table 1) showed a significant difference in the IL-10 concentration at a probability of ($P \leqslant 0.001$) between the patient group and the healthy group, as the concentration decreased significantly in the patient group (7.086±0.768) compared to the healthy group (40.891±4.463). Table 1 shows the concentration of interleukins in the patient men group (n=57) and healthy men group (n=31).

Table 1. The concentration of interleukins in the patient men group (n=57) and healthy men group (n=31).

Groups	Parameters (mean ±standard error)	
	Interleukin-8 (pg/ml)	Interleukin-10 (pg/ml)
Healthy	4.792 [a] ± 0.417	40.891 [a] ± 4.463
Patients	24.473 [b]±2.099	7.086 [b]±0.768
P-value	0.001	0.001

(a, b) letters difference means there is a significant difference.

4.2 .The concentration of interleukins (IL-8 and IL-10) and its relationship to the age groups in patients with irritable bowel syndrome

The results of the statistical analysis indicated that there were no significant differences among the age groups of patients in the concentration of IL-8 ($P > 0.05$). While the results of the statistical analysis showed that there were indeed significant differences ($P \leqslant 0.007$) between the age groups of patients in the concentration of IL-10, as the age group (41-50) years had the lowest concentration of Interleukin 10, (10.004 ± 1.775) compared with other age groups of patients (4.470 ± 0.334) and (8.032 ± 1.662) respectively.

Table 2. The relation of patient age groups (n=57) on the concentration of IL-8 and IL-10

Parameters	Categories (n=57) (mean± standard error)			P-value
	(30-40) years	(41-50) years	(51-60) years	
IL-8	21.560 [a]±2.638	23.563[a]±2.149	28.501 [a]±5.885	0.431
IL-10	10.004 [a]±1.775	4.470 [b]±0.334	8.032 [b]±1.662	0.007

(a) similar letters indicate there are no significant differences
(b) different letters indicate there are significant differences

4.3 . The concentration of interleukins (IL-8 and IL-10) and its relationship to the period of the disease in patients with irritable bowel syndrome

The test results showed that there were no significant differences in the concentration of IL-8 and IL-10 among the different period of the disease shown in Table 3**.**

Table 3: Effect of disease duration on interleukins concentration in patients with irritable bowel syndrome.

Parameters	patient (1-5) year median (lowest value - highest value)	patient (6-10) year median (lowest value - highest value)	P-value
IL-8 (pg/ml)	(12.100-97.080)19.17	(8.824-75.090)20.200	0.977
IL-10 (pg/ml)	(2.333-19.570)4.857	(1.330-26.990)4.857	0.709

5.Discussion

The statistical analysis results of the current study of patients with irritable bowel syndrome showed a significant increase in the level of IL-8 compared to the healthy group. These findings are consistent with the study of [20,21].

The current study showed that the high level of interleukin-8 in patients with irritable bowel may have a major role in the pathogenesis of the disease, as irritable bowel syndrome (IBS) was previously considered a psychosomatic disorder, but recent studies such as [22,23]. indicated that inflammation plays an important role in colon irritation, as the studies confirmed changes in Mast cell (MC), T lymphocytes and B lymphocytes, and showed higher concentrations of cytokines in the colon mucosa and in the blood of patients with Irritable Bowel Syndrome, compared to healthy subjects [24].

It has been proved that the persistent chronic inflammation has a role in the pathogenesis of the disease, as inflammatory cytokines such as IL-8 are considered one of the mechanisms causing the induction of irritable colon, as the increase in pro-inflammatory cytokines leads to damage and rupture of the small mucous membrane of the colon in most cases due to the infiltration of microbes that provoke immune responses causing mucosal and immunohistochemically disturbances, alteration in the microstructure of the mucosa and submucosa influenced by the activity of immune cells, lymphocytes, mast cells, chromaffin , enterochromaffin cells (a type of enteroendocrine and neuroendocrine cells) and antigen presenting cell [25]. The low-scale inflammation in the intestines of IBS patients is associated with activation of T lymphocytes and mast cells as well as stimulation of increased expression of lipopolysaccharides, as mast cells (MCs) and intraepithelial lymphocytes control inflammatory infiltrates in the intestine and colon of IBS patients [26].

MC activation can lead to abnormal epithelial formation in epithelial tissue cells and mucosal tissue cells, neuromuscular dysfunction, enhanced visceral hypersensitivity, and altered motility patterns of the colon which may alter enteric nerve function and motility [27]. MCs are long-lived granular cells that secrete histamine as a defense tool for colon tissue and are derived from bone marrow myeloid-cell progenitors (CD34+), but at the same time, an excess of histamine leads to a change in the function of the enteric nerve under the influence of Stem cells factor and interleukin (IL-8), which leads to increased visceral sensitivity and the occurrence of inflammation in the colon wall and may be a reason for stimulating the immune system and increasing the production of IL-8, thus raising its level [28].

Mast cells communicate with nerves in a bidirectional manner, modulating peristalsis and pain signaling, resulting in the release of proinflammatory IL-8, as MCs exert a variety of neuroprotective effects including activation of pain receptors in the cell membrane, induction of neurogenic inflammation resulting in visceral hypersensitivity; and the neuronal activation leads in turn to the release of neuropeptides and neurotransmitters, such as the 5-hydroxytryptamine receptors (5-HT); [29]. It has been established that irritable bowel syndrome patients produce high amounts of IL-8 therefore may be used as a diagnostic tool for the disease [30].Activation of the immune system plays an important role in the development of IBS symptoms when serum concentrations of pro-inflammatory cytokines (such as tumor necrosis factor alpha (TNF-α) and interleukin-17 (IL-17) and IL-8)) are increased [31]. In the normal state, the immune system of the digestive system is well adjusted to the microenvironment of the intestine and colon, resulting in a

stable homeostatic state. But the gut environment is constantly exposed to unhealthy food and microbial pathogens that help destroy this stable environment in the gut, as the balance of this stability is significantly disturbed in disease affecting the digestive system, such as IBS syndrome [32]. Colitis may be caused by immune activation upon germs entrance, therefore the nuclear factor kappa-light-chain-enhancer (NF-κB) is activated and plays an important role in maintaining a stable state of innate immunity in the gut. Dysregulation of immune responses leads to disruptions in the NF-κB pathway and to chronic inflammation [33]. Dysregulation of the NF-κB pathway -and the factors regulating it- lead to a state of uncontrolled inflammation and immune changes, as is commonly seen in colitis where levels of the pro-inflammatory cytokines IL-8 and IL-17 are increased because the NF-κB pathway enhances and activates Beta cells that produces inflammatory cytokines leading to colitis, so the level of IL-8 in the blood can be considered as a diagnostic tool for irritable bowel syndrome [34].Contaminated foodstuffs, viruses or pathogenic bacteria ingested by a person and entering his alimentary canal stimulate his immune system, and their stimulation extends to TLRs receptors on the cell surface of both immune cells (such as neutrophils and monocytes) and non-immune cells (such as epithelial cells, fibroblasts, and smooth muscle cells) as well as and toll like receptors (TLRs) in myofibroblasts under the intestinal epithelium. These stuffs can activate the innate immune system through TLR in patients with irritable bowel syndrome due to lipopolysaccharides (LPS) stimulation via TLR4 [35], [36] . TLRs occupy a key position in the first line of defense against pathogens because of their ability to recognize pathogen-associated molecular patterns. Memory T cells can express TLRs that are not found on naïve cells. Activation of inflammatory leukocytes can lead to induction or downregulation of surface expression. There is extensive regulation of intracellular signaling, and pathogens cause most of the pattern recognition receptors PRRs -after activation by pathogen molecular patterns- to upregulate the inflammatory response gene, particularly the inflammatory cytokine IL-8 in IBS[37,38]. That increases pro-inflammatory cytokines, through stimulating the immune system, such as IL-1β, IL-6, IL-8, and TNF-α for IBS patients, especially in IBS-D patients. In vitro studies also showed that the anti-inflammatory cytokine IL-10 is decreased in IBS patients and this is consistent with our current study as TLRs are activated in laboratory animals with IBS [12].

The study of [39] showed that there are specific mechanisms associated with excessive production of IL-8, IL-2, TNF-α, and decreased production of IL-10 that our current study found in colon patients, supporting the theory of imbalance between pro- and anti-inflammatory cytokines in the pathogenesis of IBS [40]. This theory reported higher expression of the chemokine-producing genes (IL-8, IL-9 and MCP 1) in IBS patients compared to healthy group [41]. In addition, IL-8 forms are more prevalent in IBS patients compared to healthy controls, which could be responsible for the lower levels of IL-10 in IBS patients [42,43].

The study of [44] showed that people with irritable colon suffer from a significant decrease of the efficiency of immune cells, especially T cells; changing from the state of normal activity to weakness and lack of responses to stimuli [45]. Therefore, their secretions and reproduction are significantly weakened, and hence causing the emergence of a chronic inflammatory state in the colon. As for the age groups and the duration of the disease, the results of the study did not show significant differences, and these results are consistent with [46] in the United States of America which included 137 IBS patients and 96 healthy subjects, divided into two age groups: 18-40 years and 48-60 years. The results did not show significant differences in the level of interleukins and age groups.Also, the current study revealed a significant decrease in the concentration of IL-10 in the group of patients with IBS, compared to the healthy group, and the results of this study were identical to those of [40,47,48]. The changes caused by intestinal inflammation may cause a decrease in interleukin (IL-10), which is produced by type 2 helper T cells (Th2) and natural killer (NKT) cells. In the normal immune system, a balance of pro- and anti-inflammatory cytokines is essential for intestinal and colonic homeostasis in blood monocytes, as intestinal monocytes and pleomorphic neutrophilic granulocytes produce large amounts of pro-inflammatory cytokines (IL-6, TNF-α, IL- 1β, IL-8) leading to differentiation and proliferation of a variety of cells and tissue damage at the expense of IL-10 production [49]. Decreased IL-10 level may lead to an imbalance between pro- and anti-inflammatory mechanisms; resulting in chronic intestinal inflammation. The

major role of IL-10 in intestinal homeostasis was revealed by genetic intervention, as IL-10 deficiency due to IL-10 receptor deficiency develops into colitis [50].

IL-10, as an anti-inflammatory cytokine, reduces the inflammatory response through a number of mechanisms and can reduce the production of inflammatory factors (including IL-1β and IFN-γ in T-cells) and reduce the activation of inflammatory macrophages [51]. The reason of the lack of IL-10 production from lymphocytes may be due to virus infection or the presence of a genetic predisposition for lack of these IL-10 cells, and thus irritability of the colon, That lack can also reduce the expression of Class II major histocompatibility complex (MHC) complex, co-stimulatory molecules and adhesion on the surface of antigen presenting cell (APCs) [52], and can prevent the growth of mass cells [53]. In our study, IL-10 levels were decreased in patients with colon disorders and this may be because pro-inflammatory cytokines were more highly expressed [54], which resulted in persistent low-grade intestinal inflammation, and antigen-presenting cells maintained their function, contributing to the adaptive immune response. Mast cell hyperplasia may be a cause of low interleukin 10 levels [55]. Since IL-10 is an immunostimulant; when it is lowered, inflammation increases and it can alter the function of the symmetric intestinal epithelial barrier [53]. Our study was consistent with that of [56] which included 38 patients with colon disease (32 females, 6 males; age range 18-70 years) and 22 healthy individuals (17 females, 5 males, age range 24-42 years) - where the diagnosis of irritable bowel syndrome was done according to the Rome criteria – and showed a decrease level of IL-10 in the blood serum of IBS patients. Inflammation and immunosuppression play an important role in the development of irritable bowel syndrome and the further development of its symptoms [57], as immunosuppression may lead to a decrease in the expression of genes related to the secretion of cytokines such as the genes responsible for IL-10 in the colonic mucosa of patients, as well as inhibiting the growth of mast cells [58]. In our study, the decreased IL-10 levels in patients with irritable colon may be due to the higher expression of the pro-inflammatory cytokine IL-8 [59] .because T cells produce proteins that are exploited to produce IL-8, at the expense of IL-10 production, which leads to persistent low-grade enteritis [60].

IL-10 plays an important role in the prevention of inflammatory and autoimmune diseases, by blocking the production of pro-inflammatory cytokines and impairing the ability of myeloid cells to efficiently activate T cells [61]. In addition, IL-10 prevents the excessive tissue damage caused by bacterial and viral infections; and further regulates and suppresses proinflammatory responses [62]. IL-10 production by T cells is a characteristic of a subset of regulatory T cells (Treg) that suppress immune responses but the production of IL-10 from these cells may decrease, because of many autoimmune diseases, which may lead to inflammation and the appearance of symptoms like IBS [63]. The low level of IL-10 in colon patients may be due to a genetic disorder and genetic defects in the IL-10 / IL-10R pathway, as they develop acute early colitis [64]. This indicates that IL-10 plays an important role in maintaining the normal immune tolerance of the gut and that its reduction causes immune disturbance and inflammation that may lead to symptoms of IBS [65].As for the comparison among age groups of people with irritable bowel syndrome, the results of the current study showed a statistically significant difference among them, as the age group (50-41) was the least significant, and this was in consistency with the study of [66,67] .The study of [66]included 108 patients with colon disorders divided into two groups of ages (18-35) and (36-55) and the duration of the disease was 6 years. The reason for this may be the abnormal immune regulation, as the presence of immunosuppression in colon patients may be caused by fatigue, sleep disturbances and the occurrence of symptoms such as anxiety and depression. These elements lead to disturbances in the pathophysiological mechanism of irritable bowel syndrome, especially fatigue. While the study of [62], which included the age group (50-41), showed that the function of the "gut-brain" axis of the central nervous system is affected by the overgrowth or imbalance of certain bacterial strains with a variety of disorders of irritable colon, visceral allergies and diseases and other autoimmune; and therefore, changes in the gut microbiota are of importance in the pathophysiology and generation of colonic symptoms. Therefore, the changes in the microbiota in the age group (50-41) of irritable bowel syndrome patients may lead to lower IL-10 compared with other age groups [62].On the other hand, androgen disturbances can act as an inflammatory modifier in men, as low testosterone levels are associated with increased metabolic risks and

systemic inflammation in IBS patients [68]as there is an inverse relationship between the level of testosterone in men and inflammatory markers in patients, and low testosterone levels have been associated with increased fat mass [57]. as testosterone is converted to estradiol in adipose tissues in the event of inflammation, thus reducing testosterone levels Furthermore, the increase in visceral fat is another cause of chronic inflammation because adipose tissues are a major source of pro-inflammatory cytokines such as IL-8, which was high in our current study [49]. This conversion of testosterone to estradiol directly inhibits the hypothalamic-pituitary axis and ultimately decreases testosterone production as visceral fat is an active secretory tissue that produces inflammatory cytokines and adipokines, thus causing a decrease in IL-10.

As for the duration of the disease, the results of our study did not show statistically significant differences for the duration of the disease for people with irritable colon, and these results were in agreement with the study of [18], which included 187 volunteers, 34.8% of whom had irritable bowel syndrome according to Rome criteria, as the duration of the disease was (1-6) years. There were no significant statistically significant differences between the duration of the disease and the level of interleukin.

6. Conclusion

Inflammation has a role in inducing irritable bowel syndrome and the age group (41-60) that was the most affected with inflammation while the duration of the disease had no relationship with Pro and anti -inflammation interleukins.

7. Acknowledgment

This work is Sponsored by Dept. of biology, College of Education for Pure Science, University of Basrah. Sincere thanks, appreciation and praise to my supervisors Dr. Hiba T. Yser, and to everyone who helped me in this research, especially my fellow masters student and Dr. Mufid k. Abu Turab and I am grateful to all the volunteer patients and wish them good health.

8. References

[1] M. Raetz, H. S-h, CL Wilhelm, D .Kirkland, A. Benson, CR. Sturge and F. Yarovinsky, Nat Immunol **14**(2), 136 (2013).
[2] E. Brian, K. Nihal and J. Patel **65**, 829 (2017).
[3] M.A.Atiyah and S.Abbas , Inter J of Psych Rehab **24**(9), 4029 (2021).
[4] S. Alatab , S. G. Sepanlou, K .Ikuta, H. Vahedi , C .Bisignano, S. Safiri and M. Naghavi, Lancet gastroentero hepatol **5**(1), 17 (2020).
[5] H.AJ.Alkatrani and M.A.Mohammad , Basrah J of Surg **28**(1), 25 (2022).
[6] A.A.Jaccob , S.Kadhim , A.malik , A.A.Mohsin and S.K.Muslim ,Pharma Assess of Gastro Comp **16**(3), 284 (2021).
[7] P. Marcos and R. Coveñas , Anatomia **1**(1), 54 (2022).
[8] PM .O'Connor , TK. Lapointe , PL. Beck and AG .Buret, Inflamm Bowel Dis **16**(6), 1411 (2010).
[9] P. E. Van den Steen, , S. J. Husson, , P. Proost, J. O. Van Damme and G. Opdenakker, Biochem Biophys Res Commun **310**(3), 889 (2003).
[10] NA .Molodecky , IS .Soon and DM.Rabi, J of Gastro **142**(1), 46 (2012).
[11] M .Bashashati, N .Rezaei, CN .Andrews ,C-Q .Chen, NE. Daryani , KA. Sharkey and M. A.Storr, Cytokine **57(**2), 9 (2010).
[12] KB .Pandey and SI .Rizvi ,Oxidative Med Cell Longev **2**(5), 270 (2009).
[13] Y. Zhen, C. Chu , S. Zhou , M. Qi and R. Shu, Mol. Med.Rep **12**(4), 5239 (2015).
[14] C. Pot, L. Apetoh, A. Awasthi and V. K. Kuchroo, J Interferon Cytokine Res **30**(6), 381 (2010).
[15] Belkaid, Y. Nat Rev Immunol **7**(11), 875 (2007).
[16] FM. Omer , JB. de Souza and EM.Riley, J Immunol **171**(10), 5430 (2003) .
[17] M. Saraiva, P. Vieira and A. O'Garra, J Exp Med **217**,1 (2020).

[18] M.J. Schmulson, D.Pulido, O.A.Rodriguez-Fandino and G.Gutierrez-Reyes,Res.esp.enferm.dig.**105**(7), 392 (2013).
[19] EJ.Vara.KA.Brokstad.T.Hausken and AG.Lied,Int J Gen Med **11**, 285 (2018).
[20] D.P.Mckerna,G.Gaszner,E.M.Quigley,J.F.Cryan and T.G.Dinan , Aliment Pharmacol.Ther **33**, 1045 (2011).
[21] A. K. Abdul Jaleel, A. F. Almulla, H. A. Alnaji, M. R. Mansor and A. Abbas Abo Algon, Iran J War Public Health **14**(2), 203 (2022).
[22] J. E. Pagán-Busigó, J. López-Carrasquillo, C. B. Appleyard and A. Torres-Reverón, PloS one, **17**(3) (2022).
[23] M. Maresca, N. Yahi, L. Younès-Sakr, M. Boyron, B. Caporiccio and J. Fantini, Toxicol Appll Pharmacol **228**(1), 84 (2008).
[24] S. Santhosh, AK .Dutta, P. Samuel, AJ.Joseph, J .Ashok Kumar and G. Kurian, Trop Gastroenterol **31**, 303 (2010).
[25] PA. Hughes, H. Zola, IA. Penttila, L A .Blackshaw, JM. Andrews and D .Krumbiegel, Am J Gastroenterol **108**, 1066 (2013).
[26] PR. Maxwell, MA. Mendall and D. Kumar, Lancet **350**(9092), 1691 (1997).
[27] L. Ohman, AC. Lindmark, S .Isaksson, I. Posserud, H. Strid and H. Sjovall, Neurogastroenterol Motil **21**(6), 644 (2009).
[28] Y. B. Tang, L. C. Yin, Y. Yang, X. H. Bo, Y. L. Cao, H. E. Wang and C. S. Lee, Acs Nano**6**(3), 1970 (2012).
[29] MR .Bhuiyan, TK. Majumder and AA .Raihan, Mymensingh Med J **19**, 275 (2010).
[30] PP. van der Veek, M .van den Berg, YE .de Kroon, HW. Verspaget and AA. Masclee, Am J Gastroenterol **100**, 2510 (2005).
[31] L. Saha, World J Gastroenterol **20**, 6759 (2014).
[32] EB .Byun, WS. Kim and NY.Sung,Cell Physiol Biochem **46**, 2072 (2018)..
[33] A. Ma and BA. Malynn, Nat Rev Immunol **12**,774 (2012).
[34] D. Zaidi, HQ. Huynh, MW. Carroll, S .Baksh and E. Wine, Clin Exp Gastroenterol **11**, 217 (2018).
[35] A .Rasquin, C .Di Lorenzo and D. Forbes, Gastroenterol **130**(5), 1527 (2006).
[36] O. Takeuchi and S. Akira, j cell **140**(6), 805 (2010).
[37] K. Takeda and S. Akira, Curr Protoco Immunol **109**(1), (2015).
[38] S L.uqman and SI .Rizvi,. Phytother Res **20**(40, 303 (2006).
[39] RM .Lovell and AC. Ford, Clin Gastroenterol Hepatol **10**(7), 712 (2012).
[40] H. D. Nguyen, H. M. Aljamaei and A. W. Stadnyk, Cell Mol Gastroenterol Hepatol **12**(4), 1343 (2021).
[41] J .Macsharry, L. O'Mahony, A. Fanning, E. Bairead, G .Sherlock and J. Tiesman, Scand J Gastroenterol **43**,1467 (2008).
[42] UC. Ghoshal, P. Abraham C. Bhatt, G. Choudhuri, SJ. Bhatia and KT. Shenoy, Indian J Gastroenterol **27**(1), 22 (2008).
[43] M .Romero Valdovinos, A. Gudiño Ramírez, J .Reyes Gordillo, WA. Martínez Flores, ME. Ramírez Miranda and P. Maravilla, Mol Biol Rep **39**, 8837 (2012).
[44] IA. Downs, OC .Aroniadis, L. Kelly and LJ .Brandt, J Clin Gastroenterol **51**, 869 (2017).
[45] WG .Thompson, KW. Heaton, GT. Smyth and C. Smyth, Gut **46**(1),78 (2000).
[46] S. Dahan, V. Busuttil, V.Imbert, J. F .Peyron,P. Rampal and D. Czerucka, Infect Immun **70**(5), 2304 (2002).
[47] S.Gregori, G. Amodio, L.Passerini and F. R. Santoni de Sio, Curr Opin Hematol **29**(4), 218 (2022).
[48] G. M. Sulaiman, R. F. Al-ansari, A. M. AL-Gebori, K. A. Khalil, S. Albukhaty, E. M. Ahmed and M. T. Soliman, Jordan J Biol Sci 15(1), (2022).
[49] W. Strober and I.J. Fuss, Gastroenterol **140**, 1756 (2011).
[50] C. Gasche, S. Bakos, C. Dejaco, W. Tillinger, S. Zakeri, W. Reinisch, J Clin Immunol **20**(5), 362 (2000).
[51] Y. Belkaid, Nat Rev Immun **7**(11), 875 (2007).

[52] T. L. Denning, N. A. Campbell, F. Song, R. P. Garofalo, G. R. Klimpel, V. E. Reyes and P. B. Ernst, Inter immun **12**(2), 133 (2000).
[53] T. Kanaya, S. Sakakibara, T. Jinnohara, M. Hachisuka, N. Tachibana, S. Hidano and H. Ohno, J Experiment Med **215**(2), 501 (2018).
[54] L .Shen, L. Su and JR. Turner , Dig Dis **27**(4), 443 (2009).
[55] AJ .Schottelius, MW .Mayo, RB. Sartor and Jr. Baldwin, J Biol Chem **274**, 31868 (1999).
[56] S. M. Bennet, A. Polster, H. Törnblom, S. Isaksson, S. Capronnier, A. Tessier and L. Öhman, J Am Gastroenterol **111**(8), 1165 (2016).
[57] H. Louis, O. Le Moine, M. Peny, E. Quertinmont, D. Fokan, M. Goldman and J. Deviere, Hepatolo **25**(6), 1382 (1997).
[58] D. Accapezzato, V. Francavilla, M. Paroli, M. Casciaro, L. V. Chircu, A. Cividini and V. Barnaba, J clin investig **113**(7), 963 (2004).
[59] K. Asadullah, W. Sterry, K.Stephanek, D. Jasulaitis, M. Leupold, H. Audring and W. D. Döcke, J clin investig **101**(4), 783 (1998).
[60] F .Driessler, K .Venstrom, R. Sabat, K .Asadullah and AJ. Schottelius, Clin Exp Immunol **135**, 64 (2004).
[61] KW .Moore, R, de Waal Malefyt, RL. Coffman and A. O'Garra, Annu Rev Immunol **19**, 683 (2001).
[62] DH .Miller and DT .Chard, O.Ciccarelli **11**, 157 (2012).
[63] W. Ouyang, S. Rutz, NK. Crellin, PA .Valdez and SG. Hymowitz, Annu Rev Immunol. Apr **23**(29), 71 (2011).
[64] J. Geginat, P. Larghi, M. Paroni, G.Nizzoli, A. Penatti, , M .Pagani and R. A. Flavell, Cytokine Growth Factor Rev **30**, 87 (2016).
[65] KH .Chang, LS. Ro, RK. Lyu and CM. Chen, Clin Chim Acta **2**(440), 64 (2015).
[66] P. Usai, R. Manca, M. A. Lai, L. Russo, M. F. Boi, I. Ibba and R. Cuomo, Eur J Intern Med **21**(4), 324 (2010).
[67] Z. Mujagic, M. Kasapi, D. M. Jonkers, I. Garcia-Perez, L. Vork, Z. Z. R. Weerts and A. A. Masclee, Gut Microbes **14**(1), 2063016 (2022).
[68] J. Bobjer, M. Katrinaki, C. Tsatsanis, Y. Lundberg Giwercman and A. Giwercman, PloS one **8**(4), e61466 (2013).
[69] M. A. Atea, S.A. Ahmed, International Journal of Psychosocial Rehabilitation **24**(9), 4029 (2020).

Journal of Basrah Researches (Sciences) **49**(1), 23 (2023)
DOI: https://doi.org/10.56714/bjrs.49.1.3

تقييم مستوى IL-8 وIL-10 في مرضى اضطراب القولون

سعاد محمد سلمان، هبه ثاقب يسر*

قسم علوم الحياة، كلية التربية للعلوم الصرفة، جامعة البصرة، البصرة، العراق.

معلومات البحث

الاستلام 18 تشرين الثاني 2022
القبول 11 اب 2023
النشر 30 حزيران 2023

الكلمات المفتاحية

انترلوكين8،انترلوكين10،IBS Human

Citation: S.M. Salman, H.T. Yser., J. Basrah Res. (Sci.) **49**(1), 23 (2023). DOI:https://doi.org/10.56714/bjrs.49.1.3

الملخص

تعد متلازمة القولون المتهيج من الاضطرابات الشائعة التي تصيب الأمعاء الغليظة. تشمل مؤشرات المرض والأعراض تقلصات وألم البطن والانتفاخ والغازات والإسهال أو الإمساك أو كليهما. صممت هذه الدراسة للتعرف على بعض المؤشرات الحيوية التي تؤثر وتتأثر بمتلازمة القولون المتهيج. هدفت الدراسة تقييم مستوى انترلوكين 8 و 10 في المرضى الذين يعانون من تهيج القولون ومقارنة مستوى هذه الانترلوكينات مع الفئات العمرية للمرضى ومدة المرض. شارك في هذه الدراسة 88 رجلا منهم 57 رجلا مصاب بمتلازمة القولون المتهيج, و31 رجلا من الاصحاء تم جمعهم من مستشفى امراض وجراحة القناة الهضمية والكبد وايضا من مستشفى الفيحاء التابعتين لدائرة صحة البصرة , قسمت عينات المرضى حسب العمر الى ثلاث فئات (30-40) و(41-50) و (51-60) سنة وقد قسمت عينات المرضى (57عينة) الى فئتين حسب الفترة الزمنية للمرض (1-5 سنوات) و (5-10 سنوات) وكانت عينات الاصحاء تتراوح اعمارهم (30-60 سنة).إذ قام الطبيب المختص بفحص المريض وإرساله الى وحدة المختبر التابعة للمركز , عندها قام الموظف المختص في المختبر بسحب ما يقارب(5 مليلتر)من الدم الوريدي الزندي vein Antecubital للحصول على مصل الدم , وقد تم قياس IL-8 و IL-10 باستخدام عدة المحاليل الجاهزة (kit) Elisa مصنع من شركة (Elabscience) وحسب رقم التسلسل (Catalog No: E-EL-H6008) لانترلوكين8 IL-8), وكذلك رقم التسلسل (Catalog No: E-EL-H6154) لانترلوكين IL-1010) وباعتماد تقنية الاليزا Linked- ELISA) . Enzymeاظهرت نتائج التحليل الاحصائي وجود ارتفاع معنوي ذو دلالة احصائية لانترلوكين 8 (IL-8) عند مستوى احتمالية P≤0.001في حين وجد انخفاض معنوي في مستوى انترلوكين 10 عند مستوى احتمالية P≤ 0.001. استنتج من الدراسة ان للالتهاب دور في استحثاث حالة تهيج القولون وان الفئة العمرية(51-60) هي الفئة الاكثر تعرضا للالتهابات في حين لم يكن لفترة المرض دور في مستوى الانترلوكينات المؤيدة والمثبطة للالتهابات.

***Corresponding author email :** heba.yser@uobasrah.edu.iq

ISSN: 1817-2695 (Print); 2411-524X (Online)
Online at: https://jou.jobrs.edu.iq