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FULL PAPER





Effect of lifestyle for Iraqi inflammatory bowel **biochemical** patients disease with some parameters

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Inflammatory bowel disease patients (IBD) are more likely to suffer from mental disorders, malnutrition and poor quality of life (QoL), all of which can result in poor clinical outcomes. The aim of this study is to find out how common malnutrition, psychiatric difficulties and poor quality of life is in IBD patients, as well as their risk factors and evaluation of the activity of Alkaline Phosphatase (ALP), Alanine Transaminase (ALT), and Aspartate Aminotransferase (AST), with measuring the level of urea and creatinine. The mean ± SD of age for the three groups (CD, UC and HC) were 27.02±7.71, 29.12±7.82 and 30.58±7.30 years respectively; ALT in the sera of both patient groups (CD&UC) was a significant decrease (p<0.05) when compared to the (HC group) while significant decrease (p<0.05) in AST in (CD group), as well as highly significant increase (p<0.001) in AST of (UC group) was discovered when compared to their levels in the matching (HC group). A highly significant increase (p<0.001) in serum ALP of (UC group) and a significant increase (p<0.05) of (CD) group in comparison to that of the (HC group), and a nonsignificant differences (p<0.05) in serum Urea and Creatinine of both patient groups (CD & UC) were discovered when compared to that of the (HC group). It was concluded that the psychological state could be one of the reasons for the occurrence of inflammatory bowel disease (IBD) which has a poor impact on QoL.

KEYWORDS

Haifaa J. Mohammed	Developing factors, inflammatory barrel disasses, ALT, ACT,
Email: haifaiabory93@gmail.com	Psychological factors; fillaminatory bower diseases; ALT; AST;
Tel.:+ 009647722806989	ALP; urea; ceratinine.

Introduction

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Inflammatory bowel diseases (IBD) are a form of chronic nonspecific Ulcerative colitis (UC) and Crohn's disease (CD) which are two gastrointestinal diseases [1]. The IBD has a complex etiology that is intimately linked to genetic predisposition, environment, microorganism and immunity [2]. Patients

with IBD are frequently plagued by humiliating gastrointestinal and systemic symptoms, which lead to an increased likelihood of hospitalization, surgery, and even mortality [3]. All of the aforementioned factors may have physical and psychological repercussions in people with IBD.

In IBD patients, anxiety and depressive symptoms are examples of psychological





disorders, discovered to be more common [4]. Furthermore, even in remission, IBD patients' nutrition is frequently affected [5] .The malnutrition has been associated to poor clinical outcomes in IBD patients. Some IBD patients have reported that certain foods have made their symptoms worse or precipitated a relapse, leading to overly restrictive diets [6], and increasing the risk of malnutrition and exacerbating nutrient insufficiency in the long run may lead to setbacks [7]. Furthermore, a study found that anxiety and depressed disorders were substantially connected with malnutrition [8]. Several studies have found that both malnutrition and psychiatric problems have the potential to considerably influence IBD patients' quality of life (QoL) [9,10]. A growing body of research reveals that a variety of combination of clinical, demographic, and psychological characteristics have been investigated as predictors of health-related quality of life (HRQoL) in patients with IBD. When it comes to demographics, IBD may be linked to a lower quality of life [11,12]. In terms of clinical variables, some research found a link between disease activity and poor healthrelated quality of life (HRQOL). Furthermore, poor medication adherence, extraintestinal symptoms, unemployment, a higher number of relapses, and surgical procedures were also observed to have a negative impact on HRQOL [13]. The impact of psychological factors on HRQOL has recently gotten a lot of attention. The study's aim is to learn more about the liver and kidney function with food and psychological state of IBD patients.

Materials and methods

The samples were taken from patients who were undergoing treatment in Gastroenterology and Hepatology Teaching Hospital in Baghdad, Iraq. Patients were invited to answer the questionnaire about the psychological state and the nature of food before, after and during the disease, as well as for healthy people to answer the nature of the food and the answers were recorded manually.

Then the samples were centrifuged at 3000xg for five minutes. The obtained serum was kept at -20 °C for the purpose of determination activity of (ALT), (AST), (ALP) and the concentrations of urea and ceratinine were measured by using Cobas C311 auto analyzer system/Switzerland.

Statistical analysis

The statistical assessment was resolved by SPSS of version 20 and the program windows, using ANOVA one-way analysis of variance. The study data were presented as mean±standerd error. The College of Science/University of Baghdad's Ethics Committee accepted this study protocol.

Results and discussion

This study includes three main groups: UC, CD, and HC. The mean ± SD ages of the three groups (CD, UC and HC) were 27.02±7.71, 29.12±7.82 and 30.58±7.30 years respectively; ALT in the sera of both patient CD and UC groups were significant decrease (p<0.05) while significant decrease (p<0.05)when compared to the (HC group) in AST in (CD group), as well as highly significant increase (p<0.001) in AST of (UC group) were observed in comparison with their levels in corresponding (HC group) as shown in Table 1. A highly significant increase (p<0.001) in serum ALP of (UC group) and a significant increase (p<0.05) of (CD) group were observed in comparison to that of the (HC group) as shown in Table 1. The results in Table 1 show a non-significant differences (p<0.05) in serum urea and ceratinine when comparing the outcomes of both patient groups (CD and UC) with (HC group).

Eurasian — Chemical Communications

Parameters	Groups			n valua
	CD	UC	НС	p-value
ALT activity (U/L)	16.26±8.06 ^{a*}	18.08±9.47 b*,c*	25.71±10.79	0.000**
AST activity (U/L)	$16.74 \pm 5.51^{a^{**}}$	87.94±32.13 ^{b**,c**}	20.20±6.38	0.000**
ALP activity (U/L)	78.33±20.73 ^{a*}	87.68±31.17 b**,c*	70.85±23.07	0.005*
Urea (mg/dL)	22.96±8.07 ^a	21.42±9.37 ^{b,c*}	26.26±7.48	0.014*
Ceratinine (mg/dL)	0.63±0.18 ^a	$0.66 \pm 0.18^{b,c}$	0.73±0.14	0.437

TABLE 1 Mean ± SD of the liver and kidney profile in serum of the three studies groups

a=Comparison between CD and HC, b= Comparison between UC and HC, c= Comparison between CD and UC,*p<0.05(Significant), **p<0.001(Highly significant), No asterisk: p>0.05 (Nonsignificant).

The patients' answers to the questionnaire showed that 95% of the population had been exposed to severe psychological trauma and affected by this shock for a period, and after a short period of this shock, the symptoms of this disease began to appear on the patients. And when they are exposed to psychological distress during the disease, the symptoms begin to increase even after taking treatment, as well as 90% of them depended on nutrition before the onset of the disease on external food and fast foods compared to healthy people. Depending on the answers of the patients under the study to the questionnaire, we noticed that psychological stress and the nature of food are among the factors causing the disease. They mentioned that, showing symptoms of the disease, they were suffering from psychological pressures such as exposure to trauma or exposure to high study pressure and other psychological and social pressures before the symptoms of the disease appear. There were previous studies that have shown that patients do suffer from oxidative stress [14-17]. With regard to food, the majority of patients' answers were that before the illness, they had been dependent on fast food for a long time.

Within the GIT, neurons and mast cells have a bidirectional contact [18], and stress can activate mucosal mast cells [19]. Mast cell activation caused by stress, which results in the production of mediators like eicosanoids, serotonin, and IL6, could play a role in IBD pathogenesis [20].

Psychological variables appear to play a role in both the pathogenesis and course of IBD, as well as how patients cope with these chronic and burdensome diseases, according to current findings. Several physical and psychological types of stress, according to Soderholm and Perdue, have an effect on various aspects of intestinal barrier function, including increased permeability of the intestine (likely due to changes in cholinergic nerve system and mast cell function in the mucosa) and promoting ions, water, mucus, and even IgA are all secreted [21]. As a result of the increased permeability, the function of the mucosal barrier is reduced, and the bacteria-host interaction is altered [22].

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Exacerbation of digestive symptoms is also linked to stress [23]. Most are aware that fast food, if consumed in excess, can harm the heart, the kidneys, and the waistlines; nevertheless, it is becoming obvious that the current diet also harms body's immunological system [24]. Diet may play a role in the condition etiology of IBD, either directly or indirectly, given the possible effects of diet on the composition and function of the microbiome in the intestine. Because of the links between the microbiome and IBD, particular dietary interventions are potentially therapeutic. Meanwhile, many IBD patients use subjective experience to judge which foods are "safe" and which are "unsafe" [25].

People with gastrointestinal illnesses, such as IBD, are more likely to develop disordered eating (DE), which is linked to greater psychological distress and symptom intensity. Long-term dieting regimens can result in maladaptive attitudes regarding eating, as well as stress [26]. While pathophysiology of clinical eating disorders



(such as anorexia nervosa is a type of anorexia) is conceivable, IBD patients are more likely to have DE, or dysfunctional eating habits such as binge eating, skipping meals, restricting, and fasting for long time without eat [27].

Dietary habits of the patient groups in this study were also indicated, where their food was free of foods that exacerbate the symptoms of disease and getting a setback, such as milk and food which are difficult to digest. We made sure that the control group was not under any psychological stress at the time of sampling, and also that their diet was balanced. The gut microbiota is a dynamic and complicated system influenced by a variety of nutrition, as well as environmental factors [28]. The microbiota is extremely important to the host's health. In fact, it inhibits the growth of harmful bacteria (such as Clostridia or Colibacillacea) in the intestine, boosts the immune system, and modulates nutritional absorption [29].

Both enzymes (AST and ALT) require pyridoxal-5'-phosphate (PLP; vitamin B6) to complete this reaction, albeit pyridoxal-5'phosphate's impact deficiency is larger on ALT activity than on AST [30]; lack of biological PLP is less likely to explain the low ALT readings in CD [31]. Hyperoxalemia may also hinder the conversion of pyridoxine to PLP in some unknown method [32]. As well, increased inflammation, TNF-, interleukin-6 (IL-6), and interleukin-8 (IL-8) the tumor necrosis factor increased oxidative damage in IBD [33] that need the treatment against these factors that the patient receives contributes to reducing them, which reduces ALT and AST [34]. The only significant predictors abnormal ALT of were immunomodulatory drugs [35]. The increase of ALP in both CD and UC may be due to the increased ALP level that was a metabolic syndrome risk factor in the middle-aged Accumulating people. data have demonstrated that the activity of increased ALP is associated with [CRP] which indicates

that serum ALP level may be a marker to reflect the low-grade chronic inflammation [36]. The (Intestinal alkaline phosphatase) (IAP) has been found to suppress the generation of proinflammatory cytokines by inhibiting NF-B activation and nuclear translocation [37], (Developmental stage, diet, and inflammation all influence the expression of IAP) [38] therefore not increase in ALP, that is, the absence of significant differences between patients for urea and creatinine means that the renal system is working well and that the biological treatment does not have a side effect on the kidneys.

According to our available information, this is the first study in Iraq in which a basic comparison was made between the two types of disease (CD&UC) and healthy people, and study of the psychological state and nutrition system on patients of IBD.

Conclusion

In conclusion, IBD patients have a significant prevalence of psychiatric illnesses and malnutrition. Our findings revealed that active disease was strongly linked to psychological disorders, nutritional risk, and poor quality of life. We also discovered that avoiding particular foods and being depressed were linked to an increased risk of malnutrition. More research is required to examine in the future, the detailed mechanisms, according to our ideas, which the psychological aspect and include psychological care as one of the most significant treatments for this condition. A decrease in ALT and AST maybe mean a decrease in B12 due to its association with it, and a rise in ALP means the body's resistance to reduce inflammation.

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References

[1] W.K. Ng, S.H. Wong, S.C. Ng, *Intest. Res.*, **2016**, *14*, 111-119. [crossref], [Google Scholar], [Publisher]

[2] D. Sgambato, A. Miranda, R. Ranaldo, A. Federico, M. Romano, *Curr. Pharm. Des.*, **2017**, *23*, 3997-4002. [crossref], [Google Scholar], [Publisher]

[3] T. Sairenji, K.L. Collins, D.V. Evans, *Prim. Care.*, **2017**, *44*, 673–692. [crossref], [Google Scholar], [Publisher]

[4] C.N. Bernstein, C.A. Hitchon, R. Walld, J.M. Bolton, J. Sareen, J.R. Walker, *Inflamm. Bowel Dis.*, **2019**, *25*, 360-368. [crossref], [Google Scholar], [Publisher]

[5] J.B. Vidarsdottir, S.E. Johannsdottir, I. Thorsdottir, E. Bjornsson, A. Ramel, *Nutr. J.*, **2015**, *15*, 61. [crossref], [Google Scholar], [Publisher]

[6] J.H. Vries, M. Dijkhuizen, P. Tap, B.J. Witteman, *Dig Dis.*, **2019**, *37*, 131-139. [crossref], [Google Scholar], [Publisher]

[7] J.K. Limdi, *Indian J. Gastroenterol.*, **2018**, 37, 284-292. [crossref], [Google Scholar],
[Publisher]

[8] G. Addolorato, E. Capristo, G.F. Stefanini,
G. Gasbarrini, *Scand. J. Gastroenterol.*, **1997**, *32*, 1013-1021. [crossref], [Google Scholar],
[Publisher]

[9] A. Tinsley, O.G. Ehrlich, C. Hwang, K. Issokson, S. Zapala, A. Weaver, G.Y. Melmed, *Inflamm. Bowel Dis.*, **2016**, *22*, 2474-2481. [crossref], [Google Scholar], [Publisher]

[10] C.K. Zhang, J. Hewett, J. Hemming, T. Grant, H. Zhao, C. Abraham, I. Oikonomou, M. Kanakia, J.H. Cho, D.D. Proctor, *Inflamm*.



Bowel Dis., **2013**, *19*, 1732–1739. [crossref], [Google Scholar], [Publisher]

[11] D. Leone, D. Gilardi, B.E. Corrò, J. Menichetti, E. Vegni, C. Correale, Allocca Mariangela, F. Furfaro, S. Bonovas, L. Peyrin-Biroulet, S. Danese, G. Fiorino, *Inflamm. Bowel Dis.*, **2019**, *25*, 1399-1407. [crossref], [Google Scholar], [Publisher]

[12] G. Velonias, G. Conway, E. Andrews, J.J. Garber, H. Khalili, V. Yajnik, A.N. Ananthakrishnan, *Inflamm Bowel Dis.*, **2017**, *23*, 283–288. [crossref], [Google Scholar], [Publisher]

[13] P.Y.M. Ho, W. Hu, Y.Y. Lee, C. Gao, Y.Z. Tan, H.H. Cheen, H.L. Wee, T.G. Lim, W.C. Ong, *Intest. Res.*, **2019**, *17*, 107-118. [crossref], [Google Scholar], [Publisher]

[14] H.A. Shaker, J.A. Zainulabdeen, N.M. ALkhalidi, *J. Glob. Pharma Technol.*, **2019**, *11*, 85-90. [<u>Pdf</u>], [<u>Publisher</u>]

[15] M. Krzystek-Korpacka, R. Kempiński, M.
A. Bromke, K. Neubauer, *Diagnostics*, **2020**, *10*, 601. [crossref], [Google Scholar],
[Publisher]

[16] M. Yuksel, I. Ates, M. Kaplan, M.F. Arikan,
Y.O. Ozin, Z.M.Y. Kilic, C. Topcuoglu, E. Kayacetin, *J. Med. Biochem.*, **2017**, *36*, 341-348. [crossref], [Google Scholar], [Publisher]

[17] A.R. Bourgonje, M. Feelisch, K.N. Faber, A. Pasch, G. Dijkstra, H. van Goor, *Trends Mol. Med.*, **2020**, *26*, 1034-1046. [crossref], [Google Scholar], [Publisher]

[18] L. Van Nassauw, D. Adriaensen, J.P. Timmermans, *Autonomic Neuroscience*, 2007, *133*, 91-103. [crossref], [Google Scholar], [Publisher]

[19] T.C. Theoharides, S. Enakuaa, N. Sismanopoulos, S. Asadi, E.C. Papadimas, A. Angelidou, K.D. Alysandratos, *Asthma & Immunology*, **2012**, *109*, 14-19. [crossref], [Google Scholar], [Publisher]

[20] T.C. Moon, A.D. Befus, M. Kulka, *Front. Immunol.*, **2014**, *5*, 569. [crossref], [Google Scholar], [Publisher]

[21] M.S. Sajadinejad, K. Asgari, H. Molavi, M. Kalantari, P. Adibi, *Gastroenterol. Res. Pract.*,



2012, 2012, Article ID 106502. [crossref], [Google Scholar], [Publisher] [22] L.C.H. Yu, J.T. Wang, S.C. Wei, Y.H. Ni, World J. Gastrointest. Pathophysiol., 2012, 3, 27-43. [crossref], Google Scholar]. [Publisher] [23] Y. Sun, L. Li, R. Xie, B. Wang, K. Jiang, H. Cao, Front. Pediatr., 2019, 7, 432. [crossref], [Google Scholar], [Publisher] [24] I.A. Myles, Nutr. J., 2014, 13, 61. [crossref], [Google Scholar], [Publisher] [25] F. Castro, H. S. de Souza, Nutrients, 2019, 11, 1398. [crossref], [Google Scholar], [Publisher] [26] T.H. Taft, S. Ballou, A. Bedell, D. Lincenberg, Gastroenterol. Clin. North Am., **2017**, 46, 847-858. [crossref], Google Scholar], [Publisher] [27] R. Satherley, R. Howard, S. Higgs, Appetite, **2015**, 84, 240-250. [crossref], [Google Scholar], [Publisher] [28] N. Hasan, H. Yang, PeerJ, 2019, 7, 7502. [crossref], [Google Scholar], [Publisher] [29] Y. Belkaid, T.W. Hand, Cell, 2014, 157, 121-141. crossref, [Google Scholar]. [Publisher] [30] R.E. Vanderlinde, Ann. Clin. Lab. Sci., **1986**, *16*, 79-93. [Pdf], [Google Scholar], [Publisher]

[31] M.R. McGill, *EXCLI journal*, **2016**, *15*, 817-828. [crossref], [Google Scholar], [Publisher]

[32] S. Vadstrup, *Scand. J. Gastroenterol.*, **2004**, *39*, 554-556. [crossref], [Google Scholar], [Publisher] [33] S. Kany, J.T. Vollrath, B. Relja, *Int. J. Mol. Sci.*, **2019**, *20*, 6008. [crossref], [Google Scholar], [Publisher]

[34] X.J. Huang, Y.K. Choi, H.S. Im, O. Yarimaga, E. Yoon, H.S. Kim, *Sensors*, **2006**, *6*, 756-782. [crossref], [Google Scholar], [Publisher]

[35] I. Parisi, J. O'Beirne, R.E. Rossi, E. Tsochatzis, P. Manousou, E. Theocharidou, M. Hamilton, C. Murray, O. Epstein, A.K. Burroughs, *Eur. J. Gastroenterol. Hepatol.*, **2016**, *28*, 786-791. [crossref], [Google Scholar], [Publisher]

[36] A. Kerner, O. Avizohar, R. Sella, P. Bartha,
O. Zinder, W. Markiewicz, D. Aronson, *Arterioscler. Thromb. Vasc. Biol.*, 2005, 25,
193-197. [crossref], [Google Scholar],
[Publisher]

[37] J. Fawley, D.M. Gourlay, *J. Surg. Res.*, **2016**, *202*, 225-234. [crossref], [Google Scholar], [Publisher]

[38] J. Bilski, A. Mazur-Bialy, D. Wojcik, J. Zahradnik-Bilska, B. Brzozowski, M. Magierowski, T. Brzozowski, *Mediators Inflamm.*, **2017**, *2017*, Article ID 9074601. [crossref], [Google Scholar], [Publisher]

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