Short Communication

Serum CXCL13 as a Potential Diagnostic Marker for Patients with Ulcerative Colitis

Mohammed Ali Qusay, Sarmad M.H. Zeiny, Haider J.M. Al-Maraashi[1]

Department of Microbiology, College of Medicine, University of Baghdad, [1]Department of Internal Medicine, Gastroenterology and Hepatology Teaching Hospital, Bab Al Muadham, Baghdad, Iraq

Abstract

Background: Ulcerative colitis (UC) is an inflammatory bowel disease that is restricted to the large intestine, characterized by superficial ulceration. The inflammation extends proximally in a contiguous manner, resulting in ulcerations, severe bleeding, toxic megacolon, and fulminant colitis. Its etiology remains unknown, and it is suggested that environmental factors influence genetically susceptible individuals, leading to the onset of the disease. CXCL13 also called B cell-attracting chemokine 1(BCA-1) it's specifically attracted B cells to inflamed site, and it's produced by Macrophages, dendritic cells and B cells. CXCL13 regulates immune response, and fibrosis in addition to the regulation of cell proliferation and apoptosis. **Objectives:** This study aims to assess the clinical significance of serum chemokine (C-X-C motif) ligand 13 as a potential diagnostic marker for identifying UC in adults. **Materials and Methods:** A case–control study was conducted with a sample size of 50 participants in the patient group and 50 healthy individuals in the control group. **Results:** This study identified a highly statistically significant difference between the patient group and the control group, indicating a very good diagnostic value for identifying UC. **Conclusion:** The outcomes of this study suggest that serum chemokine (C-X-C motif) ligand 13 can be employed as a diagnostic marker for identifying UC. It could be used as a tool for measuring disease activity in addition to the possibility of using it as a potential therapeutic target.

Keywords: Autoimmune, chemokine (C-X-C motif) ligand 13, CXCL 13, Inflammatory bowel disease (IBD), Ulcerative colitis (UC)

Introduction

Ulcerative colitis (UC) is a chronic and recurrent inflammatory bowel disease (IBD) that falls within the broader category of IBD,[1] restricted to the large intestine starting in the rectum and spreading all over the colon. The most prominent clinical manifestations of this condition are diarrhea, mucopurulent stools, and blood in the stool more systemic symptoms may also be present.[2] Symptoms vary from mild to severe during relapse which may decrease or disappear during remission,[3] and the patients suffer from recurrent remission and exacerbation.[4]

UC affects only mucosa of the colon,[5] and it can lead to complications such as bleeding ulcers, toxic megacolon, and even fulminant colitis if left untreated, the inflammation is confined to the colon and rectum, and lesions are continuous in extent from rectum and upwards to one or several colonic segments and restricted to the mucosa.[6]

The etiology of IBD is largely unknown,[7] The precise etiology of IBD remains elusive despite ongoing research efforts.[3,8-10] While epidemiological, clinical, and laboratory-based evidence all indicate that the condition is likely multifactorial, encompassing genetic, immunological, and environmental factors, its precise etiology remains complex.[11]

Studies suggest that immunological and gut microbial factors play a role in inflammatory bowel disease

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Address for correspondence: Mohammed Ali Qusay, Department of Microbiology, College of Medicine, University of Baghdad, Bab Al Muadham, 100047, Baghdad, Iraq.
Email: MohammedAliQusay@gmail.com

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(IBD)[12]. Their interactions within the intestinal mucosa trigger inflammatory responses[13]. This interaction leads to intestinal inflammation, which can be exacerbated by skewed inflammatory responses associated with genetic factors, intestinal microbiota, and environmental factors.[14] UC is linked to several extraintestinal manifestations (EIMs), which can potentially lead to more significant morbidity than the underlying intestinal condition and may even serve as the initial presentation of IBD.[15]

Cytokines

They are soluble glycoproteins with low molecular weight, in which they act in an endocrine, paracrine, or autocrine manner. The cytokine system is crucial in the body's immunological response to infection and inflammation. Immune cells produce different types of cytokines. These cytokines include chemokines, interleukins (ILs), adipokines, interferons, colony-stimulating factors, and tumor necrosis factor (TNF);[16] the development and progression of IBD are mediated by cytokines.[17]

Chemokines

Chemokines are a class of cytokines with a molecular weight of 8–10 kDa, and they play crucial roles in the human body like normal physiological functions, organ development, and immune function, and their action depends on the binding of the chemokine with its receptor to regulate immune cell interactions. Chemokines attract leukocytes (monocytes and neutrophils) from the circulation to sites of inflammation or tissue damage and activate cells to enhance wound healing or immune response.[18]

Chemokines exert their effect by interacting with a group of receptors called G-protein coupled receptors which consist of seven-transmembrane protein receptors. This results in activating different signal transduction pathways, including Janus kinase-signal transducer and activator of transcription, phosphoinositide 3-kinases, and mitogen-activated protein kinase pathway.[19]

Chemokine (C-X-C motif) ligand 13 can influence various biological functions in addition to the development of several clinical diseases.[20] CXCL13 is also called B cell-attracting chemokine 1 (BCA-1). It specifically attracts B cells, and it is an extremely essential chemokine which regulates inflammation, immune response, and fibrosis, in addition to the regulation of cell proliferation and apoptosis.[21] CXCL13 is a robust chemoattractant cytokine that encourages the movement of cells expressing its corresponding receptor, CXCR5, which is expressed on B cells as well as follicular helper CD4+ T cells (Tfh), which are expert B cell helper T cell. The interaction with antigen-presenting dendritic cells within lymph node can elicit the upregulation of CXCR5 on naive T cells, allowing them to migrate as pre-Tfh cells toward the B cell zone, where they differentiate into Tfh cells.[22]

Chemokine (C-X-C motif) ligand 13 is mainly produced by dendritic cells, monocytes, and mature macrophages. It mediates the migration of B cell to the site of inflammation which aids in the inflammatory response.[23] CXCR5 is the only receptor for CXCL13 and the interaction between them leads to stimulation of the entry of T or B cells into lymphoid organs.[20]

Materials, and Methods

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. Ethical approval for the study was obtained from the University of Baghdad, College of Medicine according to document number 1443 on November 6, 2022). It was carried out with patients verbal approval before the sample was taken.

Study design, location and timing

This case-control study was conducted between November 2022 and March 2023, to investigate the potential association between UC and a serum CXCL 13. Participants were recruited from the Gastroenterology and Hepatology Teaching Hospital at Medical City in Baghdad.

A total of 100 participants were included in the study, consisting of 50 individuals with UC, along with 50 healthy controls denoted as (Control)/(CO) for brevity. Although the groups were not sex-matched, they were matched based on age.

UC patients were evaluated under the supervision of a gastroenterologist. They were queried about the severity of their conditions using the disease activity index of UC (Truelove and Witts Severity Index). No sub-groupings were made according to disease severity due to the small numbers in the severe subgroup, making meaningful comparisons difficult. Consent was obtained, and participants provided information about their symptoms and complications, outlining risks and general information.

Apparently healthy controls were selected from the blood bank of the Gastroenterology and Hepatology Teaching Hospital, Baghdad following a comprehensive medical history assessment.

Inclusion criteria

- Male or Female patients with UC.
- Age from 18 years to 75 years.
- Patients diagnosed with only UC who do not have other autoimmune disorders.

Exclusion criteria

- Male or female patients who refuse to participate in this study.
- UC patients with other autoimmune disorders.
- Patients younger than 18.
- Patients older than 75.

Sample collection

- Venous blood samples (5 mL) were collected from pre-diagnosed UC patients after obtaining a comprehensive medical history.
- Blood samples were centrifuged at 3000 rpm for 10 min. Subsequently, the serum was placed in 1.5 mL Eppendorf tubes and stored at deep freezing (–20°C).
- The serum was then analyzed for the serum marker Hs-Crp at the International Center for Training and Development, utilizing the Enzyme-Linked Immunosorbent Assay (ELISA) technique.

Kits utilized in this study

Human CXC-chemokine ligand 13 (CXCL13) ELISA KIT Sunlong Biotech Co., LTD, Hang Zhou Shi, Yu Hang Qu, Zhe Jiang Sheng, China.

Statistical analysis

Statistical analysis was performed using Combination IBM SPSS 27 (Statistical Package for the Social Sciences, version 27, Armonk, New York, United States), and GraphPad Prism 9, San Diego, California, United States was used to perform some statistical tests and to draw figures in addition to the receiver–operating characteristic curve.

The distribution was normal concerning demographic parameters, like age and student T test where employed, but non-normal in each group regarding CXCL13 concentration. To compare the two groups, non-parametric tests, specifically the Mann–Whitney test, were employed, given the non-normal distribution. A significant difference was considered when the *P*-value was < 0.05.

Results

CXCL13 statistics

The outcomes of our investigation highlight the key findings that emerged from our comprehensive analysis. The results are presented in alignment with our research objectives, with corresponding hypotheses listed below. Demographic parameters and descriptive statistics are included, along with a comparison between the patient and control groups, revealing statistically significant differences. Additionally, the test's validity is highlighted, demonstrating excellent validity.

Demographic parameters

The target population is composed of 50 patients with UC; of whom, 23 (46%) are male patients and 27 (54%) female patients, and the control group is composed of 24 (48%) men and 26 (52%) women. The age of patients group ranged between 17 and 78 years, and for the healthy control group age was ranged between 18 and 60 years. The normally distributed data were presented as mean ± SD, the mean age of patients was 36.4 ± 10.1, and the mean age of the control group was 36.4 ± 10.6. The trends in patient groups in terms of disease duration toward less than five years, as presented in Table 1.

Descriptive statistics and comparisons between the groups

The descriptive statistics and the Mann–Whitney test results for the CXCL13 variable are summarized in Table 2. It shows the descriptive statistics for both groups including the median and percentiles of CXCL13 in the patient group and CXCL13. In the apparently healthy control group (CO), *P* value was < 0.0001 which indicates a highly significant difference between patient and (CO) groups. Figure 1 demonstrates the difference between median concentration of patients and (CO).

Table 1: Age and gender distribution between ulcerative colitis group and control group

	UC *n* = 50 (%)	CO *n* = 50 (%)
Males	23 (46%)	24 (48%)
Females	27 (54%)	26 (52%)
Age/Mean ± SD	36.4 ± 10.1	36.4 ± 10.6
Age groups		
≤40 years	35 (70%)	26 (52%)
>40 years	15 (30%)	24 (48%)
Disease duration		
≤5 years	31 (62%)	N/A
>5 years	19 (38%)	N/A

UC: ulcerative colitis, CO: control group, SD: standard deviation

Table 2: Descriptive statistics and comparison between patients and control groups

	UC (50)	CO (50)
Median pg/mL	31.53	8.647
25% Percentile	20.38	6.033
75% Percentile	48.21	13.84
Min	20.38	6.033
Max	48.21	13.84
Mann–Whitney test	P value <0.0001	

UC: ulcerative colitis, CO: control group

P value < 0.05 = significant; *P* value > 0.05 non-significant

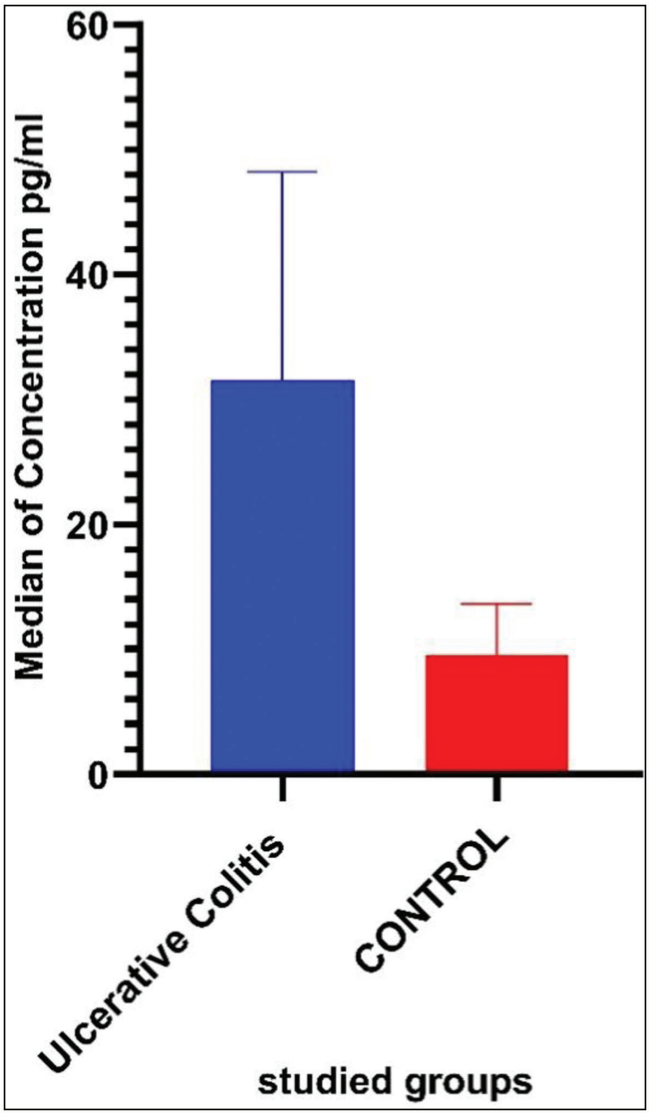

Figure 1: Median differences in levels between the patient group with ulcerative colitis and the control groups

Validity of the test

The CXCL 13 demonstrates excellent validity in the diagnosis of patients with UC, with a cut-off ≥16.8 pg/mL, and an AUC of 94.8%, signifying excellent discriminatory ability of the test. Sensitivity is 84%, specificity is 98%, positive predictive value (PPV) is 97.7%, Negative predictive value (NPV) is 86%, and accuracy is 91% ($P < 0.0001$). The test has demonstrated excellent performance, accurately distinguishing between individuals with and without the condition, as demonstrated in Figure 2.

Discussion

Throughout this study, we unraveled the multifaceted roles played by CXCL13 as a potential diagnostic marker, shedding light on its utility in diagnosing the disease.

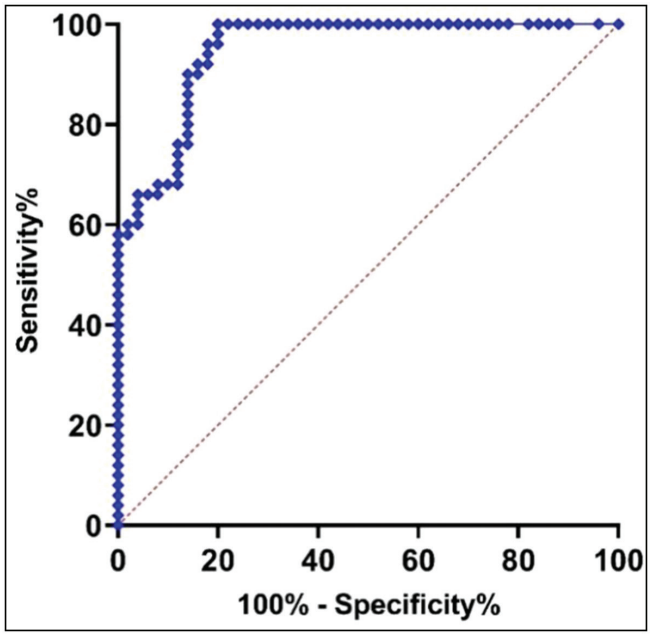

Figure 2: Receiver–operating characteristic curve analysis of CXCL13 for ulcerative colitis diagnosis

This study found that the target population possesses a higher susceptibility to UC among female patients, as demonstrated in Table 1. These findings are consistent with many different studies in Iraq regarding demographic parameters in terms of gender,[24-28].

Results of this study showed some contradiction in demographic parameters in terms of gender in KSA,[29,30] similar to the studies in Iraq such as Abdul-Hussein, Ali, Zaki, *et al.*,[12] Abdul-Hussein *et al.*,[13] and Al-Khazraji.[31]

Some studies indicate that there are no significant differences in gender distribution,[32,33].

This contradiction could be attributed to X chromosome-specific abnormalities, such as extreme inactivation skewing, inactive X chromosome reactivation, translocations, and microdeletions, which contribute to the breakdown of self-tolerance and autoimmunity,[34] X-linked genetic factors and sex hormone signaling may act together to trigger the sex-specific development of autoimmune disease.[35]

Patients with UC or Crohn's disease had increased intestinal permeability compared to healthy controls,[36] This permeability is also influenced by hormones, such as 17-beta estradiol (estrogen), prolactin, and testosterone, which are considered directly involved in symptom variation, yet their role in IBD is still poorly understood. Moreover, it is well known that the activation of estrogen receptors expressed by epithelial cells contributes to the increase of gut permeability and the activation of humoral and cellular immunity.[37]

The majority of UC cases occur in young people (17–40 years),[29] which is consistent with the results of this study as demonstrated in Table 1. The trends in disease duration observed in patients with less than 5 years of disease, as presented in Table 1, are consistent with.[31] The influence of age at diagnosis on the clinical course of UC is still controversial.[38] However, further research is needed to fully understand this relationship.

It is possible that age-related changes in the immune system, diet, family history, genetic factors, or other physiological factors, in addition to the disease phenotype, may contribute to the differences in disease extent observed in patients aged in their 40s or younger and older patients with UC as well as the disease duration. It is important to note that the Montreal classification of UC does not include age at diagnosis as a criterion, nor does the Truelove and Witts Severity Index.

The circulating levels of CXCL13 are increased in IBD patients,[39] CXCL13 plays a significant role in the pathogenesis of autoimmune disorders, cancers, and inflammatory diseases. IBD patients had a substantially higher serum CXCL13 concentration than healthy controls.[40] These findings are consistent with the result of this study. All these results suggest a good diagnostic value in this marker shedding light on its role in immune system response and function.

The researchers suggest performing more studies on this marker in larger populations, as well as subgrouping them according to severity, as mentioned before the marker exhibits very good diagnostic value with a sensitivity of 84%, specificity of 98%, PPV of 97.7%, NPV of 86%, and with an accuracy of 91%. In addition to that, this marker could be a therapeutic target for UC, due to the production of a variety of proinflammatory cytokines by B cells, including TNF-α, IL-6, IL-1β, IL-12, and IL-23. Proinflammatory cytokines produced by T follicular helper cells (Tfh cells) include IL-17, IL-21, and IL-22, but this suggestion needs more studies and research to reach this decision.

Conclusion

The serum CXCL13 possesses an excellent diagnostic value. It could be used to measure the disease activity in addition to the possibility of using it as a therapeutic target. The researchers recommend conducting further investigations on CXCL13 to explore its potential utility in combination with conventional diagnostic methods such as endoscopy, radiology, colonoscopy, and capsule endoscopy as a promising diagnostic marker for UC. These future studies should aim to evaluate whether CXCL13 can serve as a valuable tool to obviate the necessity for repetitive abdominal endoscopy-colonoscopy and capsule endoscopy procedures in assessing disease activity and pinpointing the location of inflammation.

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Conflicts of interest

There are no conflicts of interest.

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