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Expression of Dectin-1 in Patients with Acute Appendicitis: A Case-Control Study

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Abstract
Background: *Acute appendicitis is a prevalent surgical emergency characterized by inflammation of the appendix and intricate immunological reactions. Dectin-1 is a C-type lectin receptor in mammals that serves as a pattern recognition receptor, detecting fungal infections and activating several signaling pathways. Dectin-1 specifically detects β-glucans on the surface of fungal yeast cells.*
Objectives *This study aimed to estimate the concentrations of Dectin-1 in both serum and appendix tissue following appendectomy.*
Material and Methods*: A total of 100 specimens (blood and tissue) were collected from individuals aged 14 to 65 years of both sexes who underwent treatment for appendicitis at a teaching hospital in Iraq between September and December 2024. Dectin-1 concentrations were measured using enzyme-linked immunosorbent assay (ELISA).*
Results: *The mean blood Dectin-1 level in appendicitis patients was 601.21 ± 332.11 ng/L, whereas it was 588.16 ± 238.59 ng/L in the control group. There was no significant difference between the groups (P = 0.902). The average tissue concentration of Dectin-1 in appendicitis patients was 693.45 ± 300.76 ng/L, greater than the serum level, but not statistically significant (P = 0.402).* ***Conclusions:*** *These findings suggest a potential localized increase in Dectin-1 expression within the appendix tissue during inflammation, despite stable systemic levels in the bloodstream.*
Keywords: *Dectin-1, Appendicitis, Inflammatory Markers, C-type Lectin Receptors, innate immunity*

Introduction

Appendicitis, or inflammation of the vermiform appendix, is the most common surgical emergency in adolescents and young adults. Nonetheless, owing to its many clinical manifestations, recognizing this condition remains difficult even for experienced surgeons [1]. Acute appendicitis is a common cause for ED admissions, affecting approximately 70% of individuals under the age of 30 and 10% of those over the age of 60. The appendix's extensive lymphatic tissue suggests a possible involvement in the immune system [3]. The initial sign of acute appendicitis is inflammation of the appendix wall, which causes localized ischemia and, finally, perforation, that may cause either a limited abscess or widespread peritonitis [4].Proinflammatory cytokines, chemokines, and adhesion molecules are inflammatory mediators that aid in the recruitment and activation of immune cells, including neutrophils and macrophages, in the pathophysiology of acute appendicitis. These mediators are thought to have a role in the development of abscesses and perforations, as well as the clinical symptoms of appendicitis, hence affecting its outcomes [5].

Crucial for initiating natural responses, innate immune system cells include several receptors termed pattern recognition receptors (PRRs). Cells activate and initiate various responses, including phagocytosis and the release of inflammatory mediators, upon recognizing antigens via pattern recognition receptors (PRRs). The C-type lectin receptor (CLR) is a specific kind of pattern recognition receptor essential for the pathogen response. Dectin-1 operates as a type II transmembrane lectin and is a member of the C-type lectin receptor family. Dectin-1 has high expression levels in the myeloid lineage, particularly in macrophages, neutrophils, and dendritic cells (DCs). Dectin-1 identifies a range of bacterial and endogenous ligands. The activation of Dectin-1 may result in phagocytosis, the

production of reactive oxygen species (ROS), and the release of cytokines, along with other proinflammatory responses. Reactive oxygen species (ROS) are generated by mitochondrial metabolism in response to Dectin-1 ligands. Potential therapeutic and preventative targets are the signaling pathways mediated by Dectin-1. Dectin-1 markedly stimulates immune responses against several harmful pathogens, including fungus, bacteria, and parasites [7]. Dectin-1 comprises a solitary carbohydrate recognition domain (CRD) that selectively binds to β-(1→3)/(1→6) glucans, first discovered in the cell walls of particular bacteria, fungus, and other pathogens [8]. Dectin-1 is acknowledged as an extensively researched C-type lectin receptor (CTLR), exhibiting various well-defined characteristics and functioning as a prototypical receptor for signaling CTLRs. Dectin-1, although referred to as "dendritic-cell-associated C-type lectin 1," is also found on monocytes, neutrophils, and macrophages, in addition to dendritic cells [9][10]. Activation of Dectin-1 may elicit many downstream cellular responses, including the synthesis of cytokines such as TNF-α, IL-1β, IL-2, IL-8, IL-10, IL-12, and CXCL2; the initiation of phagocytosis; and a respiratory burst resulting from the formation of reactive oxygen species (ROS). Dectin-1 signaling is crucial in orchestrating adaptive immunity in conjunction with initial innate responses, promoting the activation of CD8+ T cells and the development of naïve CD4+ T cells into T-helper (Th1) or Th17 phenotypes. Dectin-1 signaling promotes appropriate adaptive and innate immune responses, as noted in numerous sources [7].

MATERIAL & METHODOLOGY

Samples collection

This study involved a total of 100 specimens, including both blood and appendix tissue samples. The participants ranged in age from 14 to 65 years and included both males and females who were diagnosed with appendicitis and underwent appendectomy between October and December 2024 at AL-Hilla Teaching Hospital, Babylon Province, Iraq.

Tissue collection

Appendix tissue samples were collected during appendectomy procedures with the assistance of surgical staff. The extraction of secretory materials from the appendix was performed based on the method described by Shnawa and Abd (2005), with some modifications [11]. Briefly, the appendix was placed in a Petri dish containing normal saline, opened longitudinally with scissors, and thoroughly rinsed with saline. The mucosal layer was scraped and suspended in 5 mL of normal saline. The suspension was centrifuged at 3500 rpm for 30 minutes, and the resulting supernatant was collected and used for the estimation of Dectin-1.

Blood collection

A total of 100 blood samples were collected from patients diagnosed with appendicitis, in addition to 50 blood samples from healthy persons constituting the control group. Five milliliters of blood were obtained by venipuncture using disposable syringes, thereafter placed into gel tubes, and centrifuged at 3000 rpm for 10 minutes. The serum was meticulously isolated, transported to Eppendorf tubes, and preserved in a deep freezer until further investigation.

Estimation of dectin-1

Dectin-1 levels in blood and tissue samples were measured using a human Dectin-1-specific enzyme-linked immunosorbent assay (ELISA) kit (BT LAB, China), following the manufacturer's instructions. A standard curve equation was used to calculate Dectin-1 levels in the samples. The statistical analysis was carried out using SPSS software (Version 23). Continuous data were reported as mean ± SD. Independent-samples t-tests were performed to evaluate serum Dectin-1 levels in patients and controls, as well as to analyze differences between serum and tissue levels. Pearson's correlation test was used to

investigate the association between Dectin-1 levels in patients' blood and tissues. A p-value of <0.05 was considered statistically significant.

Ethical approval

The Ethical Committee of the Babil Health Directorate accepted the research plan on September 15, 2024. All individuals provided verbal informed permission before sample collection. All operations involving human participants adhered to ethical norms and national legislation, under the oversight of the Ethics Committee of the Iraqi Ministry of Health.

RESULTS

In the present study, the distribution of appendicitis cases was analyzed according to age, sex, and residency. The highest proportion of cases was observed in the young adult group (20–34 years), accounting for 48% of all cases. Early adolescents (14–19 years) comprised 28% of cases, followed by middle-aged adults (35–50 years) at 16%, while the older adult group (51–65 years) represented the lowest proportion at 8%. These results are consistent with previous findings [12], which reported the highest incidence of appendicitis (39.13%) among individuals aged 20–30 years. In terms of gender distribution, 54% of patients were female and 46% were male. Figure 1 illustrates the distribution of appendicitis cases by age group, sex, and place of residence.

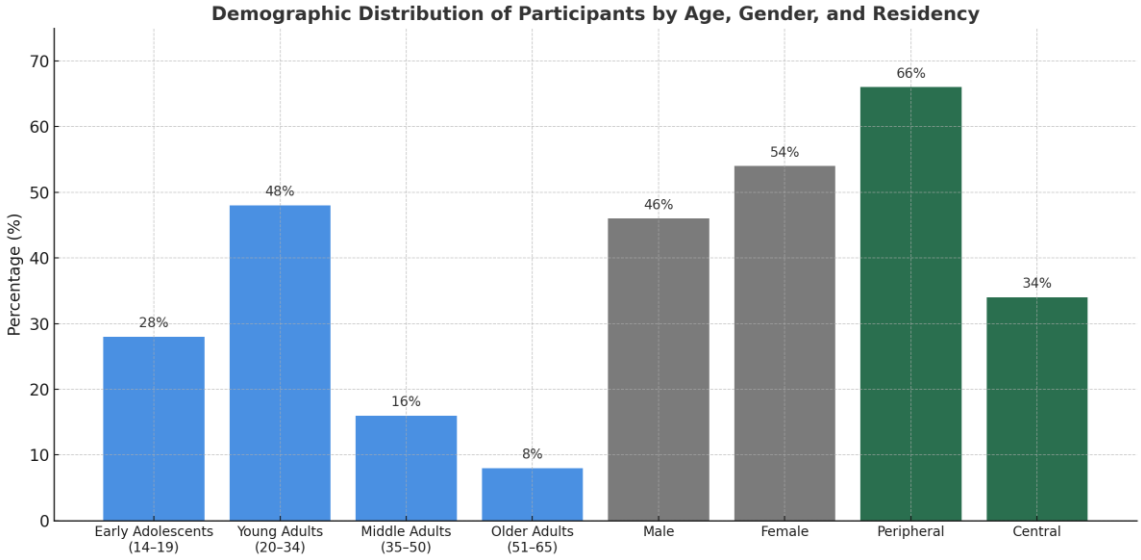

Figure 1: Distribution of the studied patients by age group, gender, and residency

To quantify Dectin-1 levels, an Enzyme-Linked Immunosorbent Assay (ELISA) kit was employed. ELISA is a highly sensitive and specific technique used for detecting and measuring target proteins, including Dectin-1, which is closely associated with immune responses, particularly in the recognition of fungal pathogens. In this study, the assessment of Dectin-1 concentrations aimed to explore its involvement in the inflammatory processes underlying appendicitis. The concentrations were determined based on the standard curve equation derived from the kit's calibration data.

The average blood concentration of Dectin-1 in appendicitis patients was 601.21 ± 332.11 ng/L, compared to 588.16 ± 238.59 ng/L in the control group. Statistical analysis indicated no significant difference between the two groups (P = 0.902), as shown in Table 1.

Table (1): Dectin-1 in serum of patient and control.

Dectin-1 serum	M±SD ng\L	P value

Patients	601.2088±332.10803	0.902 NS
Control	588.1647±238.58534	

SD: standard deviation; **NS**: not significant at P > 0.05.

The average serum Dectin-1 concentration was 601.21 ± 332.11 ng/L, while the average tissue concentration was 693.45 ± 300.75 ng/L. Statistical analysis indicated no significant difference between the two groups, shown by a P-value of 0.402, as shown in Table 2.

Table (2): Dectin-1 in serum and tissue of patient.

Dectin-1	M±SD ng\L	P value
Serum	601.21 ± 332.11	0.402 NS
Tissue	693.45 ± 300.75	

SD: standard deviation; **NS**: not significant at P > 0.05.

Pearson correlation analysis showed a weak positive correlation between serum and tissue Dectin-1 levels in patients as in table 3

Table (3) Correlation dectin-1 in serum and tissue of patient.

Dectin-1		Serum	Tissue
Serum	Pearson Correlation	1	0.250
	Sig. (2-tailed)		0.410 NS
Tissue	Pearson Correlation	0.250	1
	Sig. (2-tailed)	0.410 NS	

NS: not significant at P > 0.05.

In terms of gender comparisons, the average serum Dectin-1 levels were elevated in females (649.18 ± 413.21 ng/L) compared to males (553.24 ± 245.65 ng/L); nonetheless, this difference was not statistically significant (P = 0.581).
(P = 0.581). Tissue concentrations of Dectin-1 were elevated in females (744.66 ± 340.84 ng/L) relative to males (642.25 ± 264.84 ng/L), with no statistically significant difference (P = 0.487) as in Table 4.

Table (4) Dectin-1 in level in male and female of patients.

Parameters	Dectin-1 M±SD ng\L	P value

	Male	Female	
Serum	553.2402±245.65033	649.1778±413.21169	0.581 ^{NS}
Tissue	642.2456±264.84015	744.6600±340.84479	0.487 ^{NS}

SD: standard deviation; **NS**: not significant at P > 0.05.

DISCUSSION

The findings of the current study are consistent with those reported by Al Sehlany et al. [13] in Hilla, Iraq, who observed a higher prevalence of appendicitis in females (53.3%) compared to males (46.7%). This suggests a potential gender-related predisposition, potentially influenced by hormonal or anatomical factors. Regarding geographic distribution, patients were categorized based on their residency into central (urban) and peripheral (rural/outskirt) regions. A majority of the patients (66%) were from peripheral areas, while 34% were from central regions. In contrast to our findings a previous study conducted in Baghdad, Iraq [14], which reported a higher incidence of appendicitis in urban areas.

The current study involved the measurement of Dectin-1 concentrations in serum and appendix tissue after the appendectomy procedure. Serum levels of Dectin-1 in patients did not show a significant increase when compared to the control group. This finding indicates that the inflammatory response linked to bacterial infection and appendicitis may not have a substantial effect on systemic Dectin-1 levels. The results align with the findings of Tang et al. [15], who identified a role for Dectin-1 in the indirect regulation of gut *Lactobacilli*. Therapeutic suppression of Dectin-1 may enhance the proliferation of beneficial bacteria; however, it could also hinder the host's capacity to control opportunistic fungal infections. *Candida* species, prevalent in the human gastrointestinal tract, may enhance Dectin-1 expression; therefore, commensal fungi in the intestinal lumen should not be overlooked as potential inducers of Dectin-1 activity. The average levels of serum Dectin-1 were higher in females than in males, showing that gender did not significantly influence Dectin-1 levels in blood or tissue samples from appendicitis patients. To our knowledge, no studies have looked at Dectin-1 expression by sex in serum or appendix tissue from these patients. Al Madhoun et al. [16] looked at Dectin-1 in fat tissue and found that there were no differences in protein levels between genders, indicating that its increase during tissue inflammation does not depend on sex. Even though they were studying metabolic inflammation, their results match our finding of similar Dectin-1 levels in the appendix tissue and serum of both males and females. Notably, Dectin-1 concentrations were higher in appendix tissue compared to serum. This observation indicates that Dectin-1 may play a more localized role in mucosal immune defense, potentially contributing both to protection against fungal invasion and to the amplification of local inflammation [17] [18] [19].

Abbreviations

ED: Emergency Department
CLRs :C-type lectin receptor
DCs: dendritic cells
PRR: Pattern recognition receptors
ROS: Reactive Oxygen Species
CRD: carbohydrate-recognition domain

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