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Immunoglobulin E responses in Patients with Cutaneous and Visceral Leishmaniasis in Iraq

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A B S T R A C T

Leishmaniasis is caused by certain intracellular *Leishmania* species and is common in the tropics, where it exhibits a wide range of clinical manifestations. Both cellular and humoral immunological responses play crucial roles in disease progression. This study identified the fundamental role of B lymphocytes during the progression of leishmaniasis in human hosts. A cross-sectional study of patients with cutaneous leishmaniasis (CL) and visceral leishmaniasis (VL) from different parts of Iraq was conducted, and their respective serum IgE levels were measured before any treatment was administered. Sandwich ELISA was used for quantitative measurement of IgE in CL, VL, and control subjects. The results revealed a higher level of IgE concentration in the cutaneous patients ($p<0.05$) when compared with the healthy control group (53.21±9.1, 9.37±1.7, respectively). No significant IgE variance was observed between the visceral patients (13.2 ± 7.3) and controls. Interestingly, a significant difference ($p<0.00001$) in IgE levels was detected between the cutaneous and visceral patients. Higher IgE levels measured in patients with CL compared to those with VL may provide insight into the polarised T helper 2 immune response within active skin lesions. These findings provide vital insights into the humoral responses against the two forms of leishmaniasis.

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Introduction

Leishmaniasis is a worldwide disease caused by an intracellular parasite transmitted to humans through the bite of sand flies, primarily from the genera *Phlebotomus* and *Lutzomyia*. According to the World Health Organisation, it is a neglected disease that affects almost 350 million individuals in over 98 countries [1]. Most annual cases are reported in Western Asia, Central Asia, the Americas, and the Mediterranean Basin. In the Middle East, including Iraq, leishmaniasis is mostly endemic, where poverty, inadequate healthcare, and civil war have worsened disease propagation and elevated the exposure rate [2]. Both cutaneous (CL) and visceral (VL) types of the disease are endemic in tropical regions, with different species of the genus *Leishmania* as the causative agents of the skin (CL) or internal organs (VL) [3].

Many previous studies have investigated the complex interactions between intracellular parasites and mammalian host cells to understand the mechanisms of pathogen-mediated immune manipulation [4]. The outcome of leishmaniasis depends on the equilibrium between T helper 1 and 2 responses, in which cellular immunity is the key mediator of disease development, susceptibility, and resistance [5]. However, patients with CL exhibit stronger Th1 responses than those with VL [6]. This process involves macrophages, dendritic cells, and neutrophils [7]. Intracellular amastigotes can modify cell signalling pathways in target cells, leading to the suppression of cytokines, such as IFN-γ, and undermining the protective capacity of host cells [8]. Alternatively, the humoral response is facilitated by antibodies produced by B lymphocytes. The antibodies formed facilitate neutralisation, opsonisation, and activation of the complement system [9]. However, the humoral mechanisms underlying leishmaniasis have not been well investigated; therefore, more studies are required to elucidate these mechanisms [10].

Immunoglobulin E (IgE) is an antibody essential for mediating allergic responses, protecting against parasites [11]. It has been used as a prognostic marker in parasitic diseases such as malaria, toxoplasmosis and visceral leishmaniasis [12]. Although IgE has the lowest concentration in human serum compared to other immunoglobulins [13].

Furthermore, high levels of IgE and Th2 cytokines (IL-4, IL-5, IL-10) were observed in skin biopsies from patients with cutaneous symptoms [14]. Additionally, a consistent level of serum immunoglobulins may indicate treatment unresponsiveness and/or parasite resistance in cases of kala-azar [15]. While B cells can generate antibodies and activate antigen-presenting cells, it is essential to conduct research on certain immunoglobulins in cases of parasitic diseases [16]. Few studies have examined immunoglobulin subtype responses induced during the symptomatic phase of both cutaneous and visceral leishmaniasis [17].

In this study, the occurrence of serum IgE in patients with cutaneous and visceral leishmaniasis was investigated, with the primary intent of exploring the differences in the antibody response between the two forms of the disease, which can be utilised as a marker of infection.

Materials and Methods

Sample Population

All participants in this study were recruited from endemic areas of leishmaniasis in Iraq. Samples of cutaneous leishmaniasis were collected from Baquba Teaching Hospital in Baquba city, the centre of Diyala governorate, northeast of the capital Baghdad. The postgraduate student did not personally collect visceral leishmaniasis samples. Instead, they were submitted to the central research laboratory in Baghdad by specialist laboratory technicians working at the respective governmental hospitals across different cities in Iraq.

Sample collection was conducted from December 2024 to April 2025. Whole blood specimens of venous blood were collected in gel and clot activator tubes (AFCO, Jordan) from 44 CL and 18 VL patients attending the sampled hospitals. Serum was separated from blood by centrifugation at 3000 rpm (nuve, Turkey). For the control group, 20 blood samples were collected from healthy individuals who were clinically free of leishmaniasis. All serum samples were stored at -20 °C for later analysis.

Ethical Approval

All sample collections were approved by the Ethical Committee of the College of Science, University of Baghdad (Ref: CSEC/1124/0110) on 27 November 2024 before the commencement of the study. Before participating in the routine, and in the case of minors less than 18 years old, their parents and/or guardians signed an informed consent before they were allowed to partake in the study, according to the Declaration of Helsinki ethical guidelines for medical research relating to humans.

Experimental Design

A total of 62 random subjects of different ages, 44 with CL (25 female, 19 male) and 18 with VL (10 female, 8 male), were enrolled in this study. For patients with CL, sample acquisition was conducted under the supervision of a dermatologist at Baquba Teaching Hospital. Patients with VL were clinically diagnosed by internal medicine specialists from different hospitals across Iraq before blood samples were sent to the Central Public Health Laboratory in the capital, Baghdad. Patients with suspected CL were pre-diagnosed by a dermatologist according to the clinical appearance of observed skin ulcers. Additional direct microscopy of skin lesion samples was conducted for microscopic examination by Giemsa staining for

cutaneous leishmaniasis confirmation under oil immersion, for intracellular amastigote inspection for each skin sample, by specialist laboratory technicians working in Baquba Teaching Hospital.

Regarding patients with VL, suspected patients were primarily diagnosed by specialists according to clinical manifestations. Further diagnosis of visceral leishmaniasis was confirmed by subsequent validation using quick test cassettes (Acon, USA) and strip tests (OnSite, USA), according to the manufacturer's protocol.

Detection of serum IgE

Serum samples were analysed using an ELISA kit for IgE concentration measurement, purchased from Aviva, USA. Sandwich ELISA was performed according to the manufacturer's instructions.

Data Analysis

Statistical analysis was performed using Prism® GraphPad 8 (2019) software, where an unpaired t-test was used for the comparison of IgE concentrations between patients and healthy individuals. The predominant P-value threshold for significance was set at < 0.05.

Results

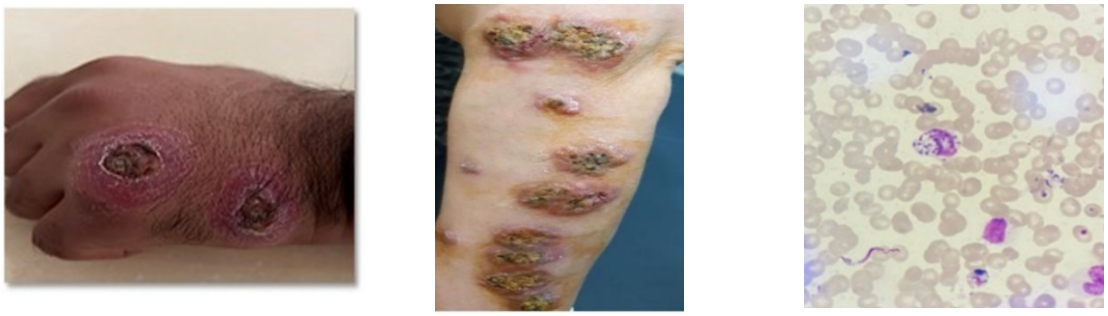
Demographic and Clinical Characteristics

In this study, 62 participants with leishmaniasis who visited local hospitals were recruited. The number of patients with cutaneous leishmaniasis was 44, comprising 25 females (56.81 %) and 19 males (43.18 %). All cutaneous leishmaniasis samples were collected from Baquba Teaching Hospital in Diyala Governorate. The number of visceral leishmaniasis samples was 18, comprising 10 females (55.56 %) and 8 males (44.44 %). The Central Public Health Laboratory in Baghdad kindly provided the VL subjects. The patients were of different ages, from 1-60 years for CL subjects, while most VL subjects were of younger ages, from less than one year to 10 years old (Table 1).

Table 1: Distribution of the age groups of CL and VL subjects sampled in their respective locations

Group	Age (yrs. Old)/No./Percentage
Cutaneous leishmaniasis (n = 44)	(1 - 18) / n=16 (36.4%)
	(19 - 50) / n= 21 (47.7%)
	(Over 50) / n= 7 (15.9%)
Visceral leishmaniasis (n = 18)	>1 / n=11 (61.1%)
	1 - 10 / n=7 (38.9%)
Total	62 patients

Ulcer scraping of each CL patient was performed in the hospital laboratory, following clinical diagnosis by a dermatologist (Figure 1). The 44 patients with cutaneous leishmaniasis exhibited a varying number of lesions across the body, affecting either the upper or lower limbs or both. A single skin lesion was detected in 24 patients (55%). Two skin lesions were observed in eight patients (18%), whereas the remaining 12 patients had multiple lesions (27%).



A B C

Figure 1: (A) Hand of a male CL patient with double lesions, (B) forearm of a female CL patient with multiple lesions, (C) amastigotes in blood smear under light microscope (1000X).

As shown in Table 2, 61.5 % of the cutaneous ulcers were found on the upper body, with the remaining 38.5 % found on the lower body. The hand was recognised as the most common location of skin ulcers on the upper body for both male and female participants. Furthermore, legs were the lowest part affected by skin lesions in both sexes. Employment and additional demographic data of the patients with CL are shown in Table 3.

Table 2: Clinical evaluation with respect to the sampled cutaneous leishmaniasis patients in the study

Site	Male	Female	Total M+F
Face	3 (6.8%)	6 (13.6%)	9 (20.4%)
Upper extremities	7 (15.9%)	10 (22.7%)	17 (38.6%)
Lower extremities	7 (15.9%)	7 (15.9%)	14 (31.8%)
Face, upper and lower extremities	1 (2.3%)	0	1 (2.3%)
Face and upper extremities	1 (2.3%)	0	1 (2.3%)
Face and lower extremities	0	1 (2.3%)	1 (2.3%)
Upper and lower extremities	0	1 (2.3%)	1 (2.3%)
Total	19 (43.2%)	25 (56.8%)	44 (100%)

Table 3: Demographic distribution of male and female participants in the study.

	Work description	Male (n=19)	Female (n=25)	Total 44 (M+F)
Employment activity for (CL)	Unemployed	0 (0%)	15 (34 %)	15 (34 %)
	Employed	7 (15.9 %)	0 (0%)	7 (15.9 %)
	Student	4 (9.1 %)	5 (11.3 %)	9 (20.45%)
	military	6 (13.6 %)	0 (0%)	6 (13.6 %)
	Child	2 (4.54 %)	5 (11.3 %)	7 (15.9%)
Residence	Urban	Male and female n=19 (43.18%)		n= 44 (100%)
	Rural	Male and female n=25 (56.82%)		

In patients with visceral leishmaniasis, fever and hepatosplenomegaly were the most common clinical manifestations observed in both male and female paediatric patients (Table 4).

Table 4: Observed symptoms of visceral leishmaniasis among the sampled patients

symptoms	Male (n=8)	Female (n=10)
Fever	8	10
Hepatomegaly	5	5
Splenomegaly	7	5
Lymph node enlargement	1	0
Jaundice	2	4
Anemia	7	9

IgE Concentration According to Age and Sex Demographic Factors in CL and VL Subjects

The concentration of total serum IgE was measured using Sandwich ELISA in 44 CL patients, 18 VL patients, and 20 healthy individuals. According to the three age groups of cutaneous leishmaniasis subjects, the result of ANOVA showed no significance between the age groups of (1-18) and (19-50) years old ($p>0.05$), whose IgE mean concentrations were

47.69±17.3 and 63.1±22.38 ng/ml, respectively. A significant difference at $p < 0.05$ was detected in the age group of seniors over 50 years old, which was 36.2±11.2 ng/ml. Additionally, no statistically significant difference in IgE mean levels in the visceral leishmaniasis subjects was observed between children below one year of age and those up to seven years old (Figure 2A).

Regarding sex, the results of both cutaneous and visceral leishmaniasis patients revealed no statistical difference ($p>0.05$) in mean of IgE concentration between males and females for all subjects, which were 52.17±24.8 and 51.3±22.6 for cutaneous leishmaniasis and also for its visceral leishmaniasis counterpart, 10.92±3.01 and 15.04±9.1, respectively (Figure 2B).

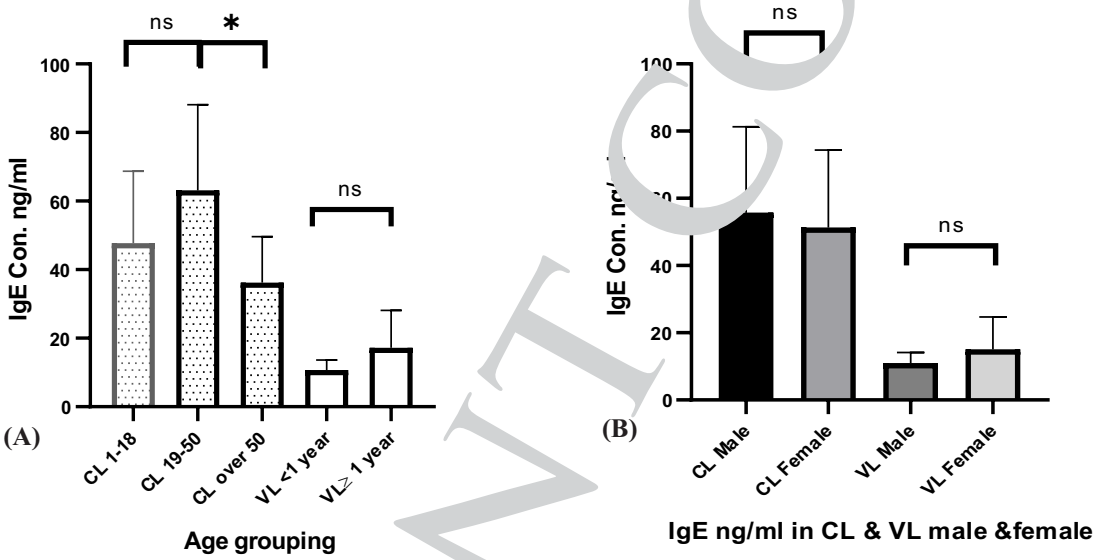


Figure 2: Mean level of IgE in the studied groups according to age (A) and sex (B), ns= non-significant, * = significant ($p < 0.05$)

Immunoglobulin E serology in CL and VL Patients

As shown in Figure 3, the mean IgE value was significantly higher ($p=0.0001$) in cutaneous leishmaniasis patients (53.21±9.1) ng/ml than in the control group (9.3±1.7) ng/ml. In contrast, paediatric visceral leishmaniasis subjects revealed only a slight non-significant increase ($p=0.058$) in the mean IgE level (13.2±7.3 ng/ml), when compared with the control.

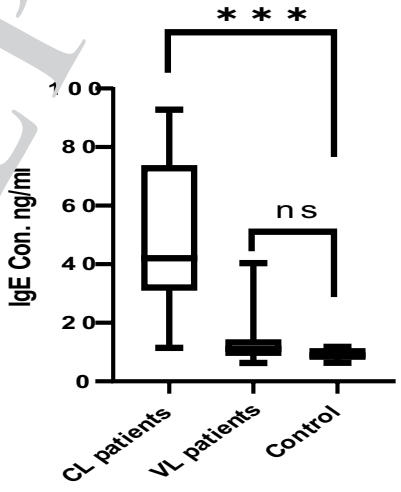


Figure 3: total serum IgE mean concentration in the studied groups (* = $p < 0.0001$, ns=non-significant)**

Discussion

The current data showed that the incidence of CL was 56.81% in females and 43.18% in males. Such a percentage of female infections is attributable to women's greater propensity to seek medical assistance compared to men [18,19]. Nonetheless, both sexes exhibit comparable susceptibility to infection risk when confronted with identical individual and behavioural characteristics [20–22]. The incidence of VL was 44.44% in males and 55.56% in females. Related studies have found that sexual dimorphism in VL was more evident in adults (post-puberty), but infection rates were similar across children. [18,23].

The cell-mediated immune response is the first line of defence against intracellular *Leishmania* spp. via macrophage phagocytic activity and cytotoxic cells [24]. Although previous studies have determined that the humoral immune response is critical in helminth infection, it is still not fully understood in the case of extracellular and intracellular protozoan parasites [25,26]. As *Leishmania* is an intracellular pathogen, B lymphocytes and their immunoglobulins have received limited attention [10]. Nevertheless, humoral immunity against leishmaniasis is controlled by the antibody production of B cells, such as potential antibodies for opsonisation and stimulation of the complement system [25]. Unsatisfactory vaccines against *Leishmania* could be attributed to non-specific antibody responses and the fact that antibody production may be linked to non-protective immune responses [27]. The IgE findings of our study are consistent with previous research, which found that elevated IgE antibodies were identified in the serum of both cutaneous and visceral leishmaniasis; they concluded that a positive link was observed between IgE levels and the range of Montenegro's reaction [28]. Furthermore, normal IgM and IgE levels could indicate treatment failure or relapse [16]; however, the increased IgE levels observed in the current study were observed in patients during the symptomatic phase of disease. Another study showed that IL-4 and IL-13 facilitate IgE secretion by B cells during cutaneous leishmaniasis, indicating poor parasite control [29,30], which supports our findings. Additionally, a high level of IgE was found to be associated with a Th2-dominant response during the progression of CL lesions, as *Leishmania* antigen can directly stimulate B cell production or indirectly through cytokine activation [31,32].

The current outcome of IgE elevation may be interpreted in light of previous research by [33], who proved that a higher IgE level was observed during the acute phase of CL and at follow-up after 6 months. In contrast, patients with CL have decreased total IgE levels after successful treatment, whereas resistant patients still have high IgE and TNF-α levels, indicating the predictive role of CL [34,35]. Interestingly, a related investigation showed that anti-leishmanial IgE was detected only in the early stages of cutaneous leishmaniasis (less than 2 months), but its counterpart, total serum IgE, was detected in early and late disease, which is in line with our results [36]. In parallel, total serum IgE levels were higher in patients with VL than in healthy individuals, indicating that non-specific IgE antibodies may be prognostic markers for protozoan and viral infections [37].

However, visceral leishmaniasis is associated with B cell activation, leading to higher total IgE levels [17]. These results are in contrast to our findings, where a non-significant mean IgE level was recorded in the studied children. This finding is likely because of the immaturity of children's immune system, which may not be capable of activating Th2 reactions as potently as in adults, and the link between IgE, T-cell suppression, and the development of disease in children is still ongoing [38,39]. A similar study in Somalia indicated no significant difference in total IgE levels between patients with VL and controls, while anti-leishmanial IgE antibodies were found only in a limited number of adult patients [40].

The findings of the current study demonstrate the difference between higher IgE levels in adult CL patients compared with normal serum IgE levels in VL patients under the age of 10 years. Such humoral IgE immunoglobulin differences could be used for CL prognosis and highlight the variance in immune response to different *Leishmania* species.

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Declarations

Authors' contributions

AHZ designed the study, statistical Analysis, supervision and follow-up, and reviewed/edited the first draft manuscript. KAS conducted the investigation and sample collection from hospitals, experimental work and wrote the first draft. All authors contributed to the development of the final manuscript and approved its submission.

Ethical Approval and Informed Consent

Written informed consent was obtained from all participants before data collection according to the Ethical Committee of the College of Science, University of Baghdad (Ref: CSEC/1124/0110) on 27 November 2024. All participants were duly informed of the objectives of the study and the protocol for sample collection. All participants signed an informed consent form. Participation was voluntary.

Disclosure of Conflict of Interest

None.

Disclosure of Funding
The study did not receive any external funding.

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