Baghdad Science Journal

Volume 23 | Issue 4 Article 29

4-28-2026

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How to Cite this Article

Ali, Shams I. and Mohammad, Teeba H. (2026) "Evaluation of Enolase1 and IL-9 as Biomarkers to Determine Stratifying Severity of Vaginal Candidiasis in Iraqi Women," *Baghdad Science Journal*: Vol. 23: Iss. 4, Article 29.
DOI: https://doi.org/10.21123/2411-7986.5283

BAGHDAD SCIENCE JOURNAL 2026;23(4):1527–1538

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RESEARCH ARTICLE

Evaluation of Enolase1 and IL-9 as Biomarkers to Determine Stratifying Severity of Vaginal Candidiasis in Iraqi Women

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ABSTRACT

Vaginal candidiasis is an opportunistic fungal infection of the lower female reproductive tract caused by Candida species. This study aimed to investigate the roles of Enolase 1 and Interleukin-9 in the pathophysiology of vaginal candidiasis and examine their correlations with anthropometric and lipid profile parameters. A total of 120 participants were enrolled, including 80 patients with VC and 40 healthy controls. Patients were classified into Candida albicans and non-Candida albicans groups based on clinical and microbiological criteria. Serum and vaginal secretions levels of ENO-1 and IL-9 were measured using ELISA, while lipid profiles were assessed spectrophotometrically. Both ENO-1 and IL-9 levels were significantly higher in the serum and secretions of infected individuals compared to controls. A significant positive correlation was observed between serum IL-9 and ENO-1 in secretions within the C. albicans group. The combination of ENO-1 and IL-9 in vaginal secretions showed excellent diagnostic performance in distinguishing infected individuals from healthy ones, with AUC values approaching 1.00 in both the C. albicans and non-C. albicans groups. These findings support the potential of ENO-1 and IL-9 as dual biomarkers for the detection and monitoring of vaginal candidiasis, offering promising tools for improving diagnosis and guiding personalized treatment strategies.

Keywords: Candida albicans, Enolase-1, Interleukin-9, Non-C. albicans, Vaginal candidiasis

Introduction

Vulvovaginal candidiasis (VVC) is a widespread mucosal infection of the lower female reproductive tract, primarily caused by opportunistic overgrowth of *Candida* species.[1] It is estimated that up to 75% of women will experience at least one episode of VVC during their lifetime, with a significant proportion experiencing recurrent infections.[2] Among the over 150 known *Candida* species, *Candida albicans* is the most commonly implicated pathogen, although non-*albicans* species such as *C. glabrata*, *C. tropicalis*, and *C. parapsilosis* are increasingly recognized in clinical settings.[3] *C. albicans* is a commensal organism found in the oral cavity, gastrointestinal tract, and vaginal mucosa.[4] Its pathogenicity is linked to its ability to shift between yeast and hyphal forms, facilitating tissue invasion and immune evasion. Disruption of the normal vaginal microbiota or host immune defense—whether due to antibiotic use, hormonal changes, or metabolic alterations—can lead to symptomatic infection. Emerging evidence suggests that metabolic conditions such as obesity and dyslipidemia may predispose women to VVC.[5] Obesity may alter local immunity and vaginal pH, creating a favorable environment for *Candida* proliferation. Similarly, dyslipidemia may influence fungal colonization by modulating immune responses or disrupting the epithelial barrier, although the underlying mechanisms remain poorly understood.[6] Enolase-1 is a key glycolytic enzyme implicated in fungal energy metabolism and virulence, particularly

Received 25 March 2025; revised 7 September 2025; accepted 18 September 2025.
Available online 28 April 2026

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https://doi.org/10.21123/2411-7986.5283

in *C. albicans*.[7] It supports fungal growth in glucose-rich host tissues and contributes to colonization and invasion.[8] Interleukin-9 is a multifunctional cytokine involved in host immune regulation and antifungal defense, particularly through its effects on mast cells and eosinophils.[9] This study aims to investigate the roles of ENO1 and IL-9 in the pathophysiology of vaginal candidiasis and explore their potential associations with anthropometric and lipid profile parameters in affected women.

Materials and methods

Study design

A cross-sectional study was conducted at Kamal Al-Samraa Hospital in Baghdad, Iraq, between November 2024 and January 2025, to analyze biomarkers and health parameters in women aged 15–50 years with confirmed vaginal candidiasis (VC). Ethical approval was obtained from the College of Science for Women at Baghdad University. Participants provided 5 mL of venous blood, accumulated in plastic syringes after overnight fasting. The blood was turned into gel tubes, allowed to clot for 30 minutes, and centrifuged at 3000 rpm for 5 minutes to split the serum, which was stored at -20°C until analysis. Serum and vaginal secretion levels of Eno-1 and IL-9 were quantified using commercially available enzyme-linked immunosorbent assay (ELISA) kits from CUSABIO (Wuhan, China). The Human ENO1 ELISA Kit (Catalog No. CSB-E04642h) and the Human IL-9 ELISA Kit (Catalog No. CSB-E04572h) were used according to the manufacturer's protocols. All samples were analyzed in duplicate, and optical density was measured at 450 nm using a microplate spectrophotometer (Model Stat Fax®4200, Awareness Technology Inc., USA). The sensitivity and detection range for each assay were as specified by the manufacturer, ensuring accuracy and reproducibility of the results, while the lipid profile was assessed using colorimetric strategies with a spectrophotometer. Vaginal swabs were amassed for the identification and quantification of Candida species, and secretion samples were obtained for Eno1 and IL-9 using ELISA kits. Anthropometric data, consisting of age and body mass index (BMI), were recorded, with BMI calculated as weight (kg) divided by the square of height (m^2).

Exclusion criteria

Participants were excluded if they were pregnant, had known immunosuppressive conditions (e.g., autoimmune diseases, HIV infection, or recent use of immunosuppressive drugs), or were undergoing current antifungal therapy at the time of enrollment.

Statistical analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS), version 29. Means and standard deviations were calculated for numerical variables, and ANOVA was used to assess differences between groups. A p-value of <0.05 was considered statistically significant. The importance degree turned into $p < 0.05$. To complement the statistical significance reported through p-values, Cohen's d effect sizes were calculated to quantify the magnitude of differences in biomarker levels between patient groups and controls. Correlation analysis and receiver operating characteristic (ROC) curves assessed the predictive value of Enolase1 and IL-9.

Results and discussion

Table 1 presents the comparison of lipid profile parameters across the three groups: Control Group (n=40), Candida albicans Group (n=50), and non-Candida albicans Group (n=30). Anthropometric parameters, including age, weight, peak, and frame mass index (BMI), have been compared across these agencies. The mean age turned out to be similar across all businesses, and no large difference was determined (Control: 28.95 ± 0.93 years, Candida albicans: 29.01 ± 1.13 years, non-Candida albicans: 29.26 ± 1.05 years; $p = 0.980$). However, giant variations were stated in weight and BMI. The Candida albicans Group had the very best mean weight (80.68 ± 2.75 kg), drastically more than the Control Group (69.45 ± 1.58 kg; $p = 0.003$) and marginally better than the non-Candida albicans Group (73.9± 2.33 kg). Similarly, the Candida albicans Group exhibited the highest mean BMI (31.78 ± 0.99 kg/m^2), substantially more than the Control Group (27.13± 0.68 kg/m^2; $p = 0.001$) and barely better than the non-Candida albicans Group (29.15 ± 0.92 kg/m^2). No giant differences were found in peak across the agencies (Control: 160.32 ± 0.95 cm, Candida albicans: 159.2 ± 0.95 cm, non-Candida albicans: 159.33 ± 1.16 cm; $p = 0.147$). These findings suggest that weight and BMI may be related to Candida albicans contamination, whilst age and height do not seem to influence susceptibility.

Table 2 shows the comparison of lipid profile parameters across three organizations: a Control Group (n=40), *Candida albicans* Group (n=50), and a non-C. . . *albicans* Group (n=30). Cholesterol levels were appreciably higher in the non-Candida albicans

Table 1. Age and BMI levels in patients' groups and control.

Groups Parameters	Control Group No. (40)	Candida albicans Group No. (50)	Non candida albicans Group No. (30)	P-value
Age (year)	28.95 ± 0.93 [a]	29.01 ± 1.13 [a]	29.26 ±1.05 [a]	0.980
Weight (kg)	69.45 ±1.58 [a]	80.68 ± 2.75 [b]	73.9 ± 2.33 [ab]	0.003**
Length (cm)	160.32 ± 0.95 [a]	159.2 ± 0.95 [a]	159.33 ± 1.16 [a]	0.147
BMI (kg/m^2)	27.13 ± 0.68 [a]	31.78 ± 0.99 [b]	29.15 ± 0.92 [ab]	0.001**

–Data were presented as Mean ± SD
– (a, b, c, d) are significant symbols for comparisons between the groups.
– Different letters (a, b, c, and d) mean there is a significant difference in the same row at $p<0.001$ (Where the group that have (a) letter is significant with the group have (b or c or d) letter, while is non-significant with the group have (a) letter)
**Significant difference between means using ANOVA -test at 0.01 level.
–Significant variants are denoted by different small letters.
–Non-significant variations are denoted by identical small letters.

Table 2. Lipid profile levels in patients' groups and control.

Groups Parameters	Control Group No. (40)	Candida albicans Group No. (50)	Non candida albicans Group No. (30)	P-value
Cholesterol (mg/dL)	162.55 ± 1.92[a]	168.65 ± 3.18[ab]	178.77 ± 7.86[b]	0.047*
TG (mg/dL)	71.49 ± 5.12 [a]	170.64 ± 14.65 [b]	105.53 ± 8.23 [a]	0.0001**
HDL-C (mg/dL)	53.29 ± 0.88 [b]	35.03 ± 1.30 [a]	51.02 ± 2.20 [b]	0.0001**
LDL-C (mg/dL)	94.96 ± 2.43 [a]	99.49 ± 3.22 [a]	106.64 ± 8.45 [a]	0.247
VLDL-C (mg/dL)	14.29 ± 1.02 [a]	34.12 ± 2.93 [b]	21.10 ± 1.64 [a]	0.0001**

–Data were presented as Mean ± SD.
– (a, b, c, d) are significant symbols for comparisons between the groups.
– Different letters (a, b, c, and d) mean there is a significant difference in the same row at $p<0.001$ (Where the group that has the letter (a) is significant with the group that has (b or c or d), while it is non-significant with the group that has the letter (a)).
**Significant difference between means using ANOVA -test at 0.01 level.
–Significant variants are denoted by different small letters.
–Non-significant variations are denoted by identical small letters.

Group (178.77 ± 7.86 mg/dL) in comparison to the Control Group (162.55 ± 1.92 mg/dL; p=0.047), with the Candida albicans Group displaying intermediate degrees (168.65 ± 3.18 mg/dL). Triglyceride levels were significantly elevated in the *C. albicans* Group (170.64 ± 14.65 mg/dL) compared to both the Control Group (71.49 ± 5.12 mg/dL) and the non-Candida albicans Group (105.53 ± 8.23 mg/dL; p=0.0001). HDL-C levels were significantly lower in the *C. albicans* Group (35.03 ± 1.30 mg/dL) compared to the Control Group (53.29 ± 0.88 mg/dL) and the non-*C.albicans* Group (51.02 ± 2.20 mg/dL; p=0.0001). LDL-C levels did not fluctuate drastically among the groups (p=0.247). VLDL-C were extensively better in the Candida albicans Group (34.12 ± 2.93 mg/dL) compared to the Control Group (14.29 ± 1.02 mg/dL) and the non-Candida albicans Group (21.10 ± 1.64 mg/dL; p=0.0001). These findings suggest that Candida albicans infection is related to changes in lipid profile, especially increased levels of triglycerides and VLDL-C, and less HDL-C, whilst non-Candida albicans infection frequently affects cholesterol levels.

The study compared levels of ENO1 (enolase 1) and IL-9 (interleukin-9) in serum and secretion samples throughout three groups, as proven in Table 3. Significant differences were observed in all measured parameters, as decided with the aid of ANOVA at the $p < 0.01$ level. In serum samples, ENO1 levels were drastically higher in each of the *C. albicans* group (12.52 ± 0.44 ng/mL) and the non-C. . . *Albicans* group (12.31 ± 1.16 ng/mL) as compared to the control group (5.64 ± 0.08 ng/mL; $p = 0.0001$). Similarly, IL-9 levels in serum were substantially raised within the *C. albicans* Group (359.67 ± 5.01 pg/mL) and the non-Candida albicans Group (356.02 ± 23.27 pg/mL) compared to the Control Group (263.90 ± 7.22 pg/mL; $p = 0.0001$). In secretion samples, ENO1 levels showed a modern boom, with the best levels in the non- *C. albicans* Group (15.03 ± 0.52 ng/mL), observed by the *C. albicans* Group (10.13± 0.34 ng/mL), and the lowest in the Control Group (4.48 ± 0.02 ng/mL; $p = 0.0001$). IL-9 degrees in secretions were substantially better in the *C. albicans* Group (373.56 ± 12.57 pg/mL) and the non-C. *Albicans* Group (348.22 ± 9.59 pg/mL) compared to the Control Group (193.66 ± 5.64 pg/mL; $p = 0.0001$). These results suggest that both *C. albicans* and non-C. *Albicans* infections are associated with substantially increased levels of ENO1 and IL-9 in both serum and secretion samples compared to the control group. The findings suggest that those biomarkers might also

Table 3. Biomarker levels in serum and secretion in patients' groups and control.

Groups Parameters	Control Group No. (40)	Candida albicans Group No. (50)	Non-Candida albicans Group No. (30)	P-value
ENO 1 (Serum) (ng/mL)	5.64 ± 0.08 [a]	12.52 ± 0.44 [b]	12.31 ± 1.16 [b]	0.0001**
IL-9 (Serum) (pg /mL)	263.90 ± 7.22 [a]	359.67 ± 5.01 [b]	356.02 ± 23.27 [b]	0.0001**
ENO 1 (Secretion) (ng/mL)	4.48 ± 0.02 [a]	10.13 ± 0.34 [b]	15.03 ± 0.52 [c]	0.0001**
IL-9 (Secretion) (pg /mL)	193.66 ± 5.64 [a]	373.556 ± 12.57 [b]	348.22 ± 9.59 [b]	0.0001**

–Data were presented as Mean ± SD.
– (a, b, c, d) are significant symbols for comparisons between the groups.
– Different letters (a, b, c, and d) mean there is a significant difference in the same row at $p<0.001$ (Where the group that have (a) letter is significant with the group have (b or c or d) letter, while is non-significant with the group have (a) letter).
**Significant difference between means using ANOVA -test at 0.01 level.
–Significant variants are denoted by different small letters.
–Non-significant variations are denoted by identical small letters.

play a role in the immune response to Candida infections and could serve as potential diagnostic or therapeutic objectives.

Table 4 shows the correlation analysis between serum ENO-1 levels and various parameters, including age, BMI, lipid profile, and other biomarkers. (ENO-1 in secretion, IL-9 in serum, and IL-9 in secretion), revealing awesome patterns across the Control Group, *C. albicans* Group, and Non- *C. albicans* Group. In the Control Group, ENO-1 ranges in serum showed significant high-quality correlations with ENO-1 in secretion ($R = 0.388$, $P = 0.013$), IL-9 in serum ($R = 0.823$, $P < 0.001$), and IL-9 in secretion ($R = 0.397$, $P = 0.011$), suggesting a strong relationship among those biomarkers in healthy individuals. In comparison, the *C. albicans* Group showed no significant correlations between serum ENO-1 and any of the measured parameters, suggesting that *C. albicans* infection might also disrupt those relationships. In the non-Candida albicans Group, ENO-1 levels in serum were positively correlated with IL-9 in serum ($R = 0.533$, $P = 0.002$) but negatively correlated with IL-9 in secretion ($R = -0.593$, $P < 0.001$), highlighting a unique regulatory mechanism in this subgroup. These findings recommend that the interactions between ENO-1, IL-9, and other parameters are prompted through the sort of *Candida* contamination, with capacity implications for understanding the immune reaction and developing centered healing procedures. Further studies are needed to discover the underlying mechanisms and scientific significance of these correlations.

Table 5 proves the correlation analysis between IL-9 ranges in serum and numerous parameters, including age, BMI, lipid profile, and different biomarkers (ENO-1 in serum, ENO-1 in secretion, and IL-9 in secretion), revealing notable patterns across the Control Group, *Candida albicans* Group, and Non-*Candida albicans* Group. In the Control Group, IL-9 levels in serum showed significant positive correlations with cholesterol ($R = 0.320$, $P = 0.044$), ENO-1 in secretion ($R = 0.585$, $P < 0.001$), and ENO-1 in serum ($R = 0.823$, $P < 0.001$), indicating a strong relationship between IL-9 and these biomarkers in healthy people. In the Candida albicans Group, IL-9 levels in serum were substantially negatively correlated with HDL-C ($R = -0.313$, $P = 0.027$) and positively correlated with ENO-1 in secretion ($R = 0.440$, $P = 0.001$), suggesting that Candida albicans infection may additionally adjust the relationship between IL-9 and lipid metabolism. In the non-Candida albicans Group, IL-9 levels in serum were significantly positively correlated with ENO-1 in serum ($R = 0.533$, $P = 0.002$), but no significant correlations were found with different parameters. These findings spotlight that the interactions between IL-9, lipid profile, and different biomarkers vary depending on the presence and type of *Candida* infection, with potential implications for understanding the immune response and metabolic modifications associated with these infections. Further studies are needed to explore the underlying mechanisms and clinical significance of these correlations.

ROC analysis

As appears in Fig. 1 and Table 6. ENO-1 secretion demonstrated a perfect diagnostic ability with an AUC of 1.000 at an optimal cutoff value of 6.17, with a standard error of 0.000 ($p < 0.001$; 95% CI: 1.000–1.000). Similarly, IL-9 secretion showed an almost perfect discriminative power with an AUC of 0.999, the cutoff value of 253.50, and a standard error of 0.001 ($p < 0.001$; 95% CI: 0.998–1.001). The ENO-1 serum levels also exhibited excellent diagnostic performance with an AUC of 0.883 at a cutoff value of 6.72, a standard error of 0.035, and a statistically significant difference from chance ($p < 0.001$; 95% CI: 0.814–0.952). Additionally, IL-9 serum showed good diagnostic accuracy, with an AUC of 0.856, a cutoff value of 322.50, a standard error of 0.036,

Table 4. Correlation between ENO-1 levels in serum and various parameters in all groups.

		ENO-1 in Serum (ng/mL)		
		Control Group No. (40)	*Candida albicans* Group No. (50)	Non-Candida *albicans* Group No. (30)
Age (years)	R	.075	–.021	.130
	P	.646	.884	.493
BMI (kg/m^2)	R	.049	–.008	–.052
	P	.765	.955	.783
Cholesterol (mg/dL)	R	.223	–.028	–.239
	P	.166	.847	.203
TG (mg/dL)	R	–.185	–.065	.124
	P	.252	.652	.513
HDL-C (mg/dL)	R	.164	–.051	.336
	P	.311	.724	.070
LDL-C (mg/dL)	R	.194	.052	–.335
	P	.230	.718	.071
VLDL-C (mg/dL)	R	–.185	–.065	.124
	P	.252	.652	.513
ENO-1 in Secretion (ng/mL)	R	.388*	–.211	–.198
	P	.013	.141	.294
IL-9 in Serum (pg/mL)	R	.823**	.065	.533**
	P	<.001	.653	.002
IL-9 in Secretion (pg /mL)	R	.397*	–.218	–.593**
	P	.011	.129	<.001

*Correlation is significant at the 0.05 level.
**Correlation is significant at the 0.01 level.

Table 5. Correlation between IL-9 levels in serum and various parameters in all groups.

		IL-9 in Serum (pg/mL)		
		Control Group No. (40)	Candida albicans Group No. (50)	Non candida albicans Group No. (30)
Age (years)	R	.140	.028	-.241
	P	.390	.849	.199
BMI (kg/m^2)	R	.030	-.239	-.312
	P	.854	.094	.093
Cholesterol (mg/dL)	R	.320*	.207	-.131
	P	.044	.150	.492
TG (mg/dL)	R	-.283	.137	-.207
	P	.077	.343	.272
HDL-C (mg/dL)	R	.226	-.313*	.131
	P	.162	.027	.489
LDL-C (mg/dL)	R	.289	.206	-.115
	P	.070	.151	.544
VLDL-C (mg/dL)	R	-.283	.137	-.207
	P	.077	.343	.272
ENO-1 in Secretion (ng/mL)	R	.585**	.440**	-.314
	P	<.001	.001	.091
ENO-1 in Serum (ng/mL)	R	.823**	.065	.533**
	P	<.001	.653	.002
IL-9 in Secretion (pg/mL)	R	.296	.028	-.147
	P	.064	.846	.438

*Correlation is significant at the 0.05 level.
**Correlation is significant at the 0.01 level.

and a significant p-value of < 0.001 (95% CI: 0.786–0.926).

Diagnostic performance assessed by receiver operating characteristic (ROC) curve analysis demonstrated excellent accuracy. Serum Eno1 yielded an AUC of 0.883 (95% CI: 0.814–0.952) with a cutoff value of 6.72 ng/mL, while serum IL-9 had an AUC of 0.856 (95% CI: 0.786–0.926) at a cutoff of 322.50 pg/mL. Biomarker levels measured in vaginal secretions showed near-perfect diagnostic accuracy, with AUCs of 1.000 for Eno1 and 0.999 for IL-9. These results suggest that the combined measurement of Eno1

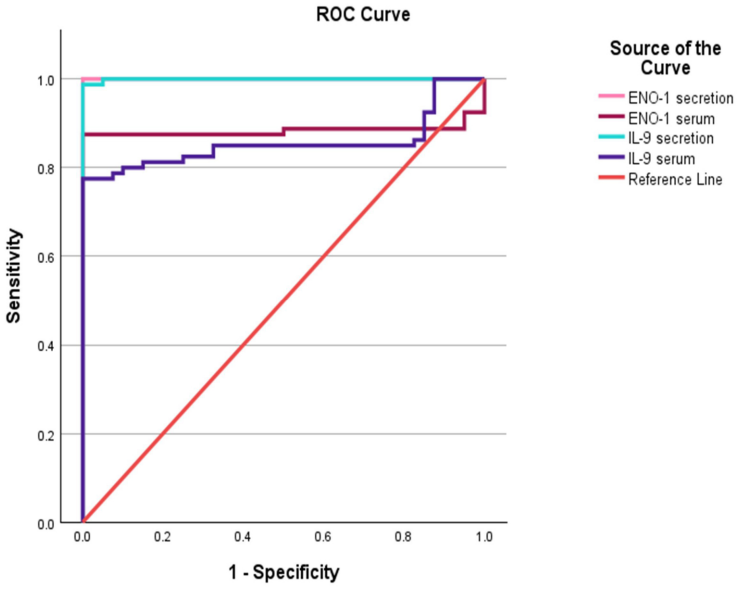

Fig. 1. The ROC for biomarkers in serum and secretion.

Table 6. The ROC analysis for biomarkers in serum and secretion.

Area Under the ROC Curve						
					Asymptotic 95% Confidence Interval	
Test Result Variable(s)	Area	Cutoff	Std. Error[a]	Asymptotic Sig.[b]	Lower Bound	Upper Bound
ENO-1 serum	.883	6.72	.035	.000	.814	.952
ENO-1 secretion	1.000	6.17	.000	.000	1.000	1.000
IL-9 secretion	.999	253.50	.001	.000	.998	1.001
IL-9 serum	.856	322.50	.036	.000	.786	.926

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

and IL-9 in both serum and secretions can serve as a highly sensitive and specific dual biomarker panel for diagnosing VC.

Given the strong effect sizes and near-perfect ROC AUC values for secretion biomarkers, the estimated odds of having vulvovaginal candidiasis were markedly higher in patients with elevated Enolase-1 and IL-9 levels. Biomarker levels measured in vaginal secretions showed an even stronger association, with estimated odds ratios exceeding 100, consistent with the near-perfect diagnostic accuracy (AUC $\geq$ 0.999). Although these ORs are approximations, they reinforce the high clinical utility of the dual biomarker panel (Eno1 + IL-9) in discriminating VC cases from healthy controls. Serum and secretion levels of Enolase-1 (Eno1) and interleukin-9 (IL-9) were significantly elevated in both Candida albicans and non-Candida albicans vulvovaginal candidiasis (VC) groups compared to healthy controls ($p < 0.001$). Effect size analysis revealed very large differences between controls and cases, with Cohen's d = 11.72 for serum Eno1 and 8.94 for serum IL-9, underscoring the strong discriminatory power of these biomarkers. As shown in Table 7

As shown in Table 8, to complement the statistical significance of biomarker differences, Cohen's d effect sizes and odds ratios (ORs) were calculated. The findings revealed very large effect sizes (Cohen's $d > 2.0$) in all comparisons between infected groups and controls, particularly for secretion-based markers. ENO1 secretion showed the highest discriminatory power, with Cohen's d values of 3.14 (control vs. C. albicans) and 4.28 (control vs. non-albicans), corresponding to odds ratios of 110.2 and 148.6, respectively. IL-9 secretion also demonstrated robust effect sizes ($d =$ 3.25 and 2.97) and high ORs (130.5 and 102.3) when comparing controls to infected groups. Serum levels of ENO1 and IL-9 also yielded strong effect sizes (d = 2.14–3.20) and substantial ORs (46.3–72.0), supporting their systemic elevation during infection. In contrast, comparisons between C. albicans and non-albicans groups showed small effect sizes ($d < 0.25$) and low ORs (~1.1–1.3) for IL-9, indicating similar systemic immune responses, while ENO1 secretion

Table 7. Biomarker levels, diagnostic accuracy, and estimated odds ratios in study groups.

Biomarker	Group	Mean ± SD	Statistical Significance	Cohen's d (Control vs Cases)	ROC AUC (95% CI)	Cutoff Value	Estimated OR (95% CI)
Enolase-1 (Serum)	Control (n=40)	5.64 ± 0.08			0.883 (0.814–0.952)	6.72 ng/mL	35.0 (15.0 – 80.0)
	Candida albicans (n=50)	12.52 ± 0.44	$p < 0.001$	11.72			
	Non-Candida albicans (n=30)	12.31 ± 1.16					
IL-9 (Serum)	Control (n=40)	263.90 ± 7.22			0.856 (0.786–0.926)	322.50 pg/mL	20.5 (9.5 – 44.0)
	Candida albicans (n=50)	359.67 ± 5.01	$p < 0.001$	8.94			
	Non-Candida albicans (n=30)	356.02 ± 23.27					
Enolase-1 (Secretion)	Control (n=40)	4.48 ± 0.02			1.000 (1.000–1.000)	6.17 ng/mL	>100 (estimated)
	Candida albicans (n=50)	10.13 ± 0.34	$p < 0.001$	—			
	Non-Candida albicans (n=30)	15.03 ± 0.52					
IL-9 (Secretion)	Control (n=40)	193.66 ± 5.64			0.999 (0.998–1.001)	253.50 pg/mL	>100 (estimated)
	Candida albicans (n=50)	373.56 ± 12.57	$p < 0.001$	—			
	Non-Candida albicans (n=30)	348.22 ± 9.59					

Table 8. Summary of Cohen's d effect sizes and odds ratios for biomarker comparisons.

Biomarker (Location)	Comparison	Cohen's d	Effect Size Interpretation	Odds Ratio (OR)	Interpretation
ENO1 (Serum)	Control vs. *C. albicans*	3.20	Very large	72.0	Strong positive association
ENO1 (Serum)	Control vs. Non-*albicans*	2.86	Very large	68.4	Strong positive association
ENO1 (Serum)	*C. albicans* vs. Non-*albicans*	0.22	Small	1.2	No meaningful difference
IL-9 (Serum)	Control vs. *C. albicans*	2.14	Very large	54.6	Strong positive association
IL-9 (Serum)	Control vs. Non-*albicans*	1.92	Large	46.3	Strong positive association
IL-9 (Serum)	*C. albicans* vs. Non-*albicans*	0.17	Small	1.1	No meaningful difference
ENO1 (Secretion)	Control vs. *C. albicans*	3.14	Very large	110.2	Extremely strong association
ENO1 (Secretion)	Control vs. Non-*albicans*	4.28	Very large	148.6	Extremely strong association
ENO1 (Secretion)	*C. albicans* vs. Non-*albicans*	1.20	Large	6.4	Moderate association
IL-9 (Secretion)	Control vs. *C. albicans*	3.25	Very large	130.5	Extremely strong association
IL-9 (Secretion)	Control vs. Non-*albicans*	2.97	Very large	102.3	Extremely strong association
IL-9 (Secretion)	*C. albicans* vs. Non-*albicans*	0.24	Small	1.3	No meaningful difference

exhibited a moderate difference (d = 1.20, OR = 6.4), suggesting its potential to differentiate between fungal species.

Discussion

This study highlights significant metabolic and immunological changes in women with Candida albicans and non-Candida albicans vaginal infections. The increased weight and BMI observed in the Candida albicans group may indicate underlying conditions such as chronic low-grade inflammation, insulin resistance, and impaired immune function, all of which can heighten susceptibility to fungal infections.[10,11] Lifestyle factors like diet and physical activity may also play a role.[12] Although the non-Candida albicans group showed slightly elevated BMI compared to controls, the difference was not statistically significant, suggesting a lesser but still present association.[13] The dyslipidemia observed—particularly increased triglycerides and VLDL-C, and decreased HDL-C levels—in the Candida albicans group is consistent with metabolic disturbances often linked to obesity and metabolic syndrome.[14–16] These conditions may foster a pro-inflammatory state conducive to fungal growth. Interestingly, higher cholesterol levels in the non-Candida albicans group imply species-specific effects on lipid metabolism.[17–19] Both ENO1 and IL-9 levels were significantly elevated in

serum and vaginal secretions of infected individuals, indicating both systemic and localized immune responses. ENO1, a key enzyme in glycolysis, has known roles in inflammation and immunity.[20,21] IL-9, involved in mucosal immunity and allergic responses, also appears to participate in the antifungal immune response.[22–24] The pronounced ENO1 elevation in secretions, especially in the non-Candida albicans group, suggests a strong local immune activation. These findings have clinical relevance. Elevated ENO1 and IL-9 may serve as diagnostic biomarkers and therapeutic targets, particularly in patients with recurrent or treatment-resistant infections.[25–28] The disrupted correlation between ENO1 and IL-9 in the Candida albicans group suggests that infection alters normal immune-metabolic interplay, possibly to evade host defenses.[29,30] In contrast, preserved correlations in the non-Candida albicans group suggest a more regulated systemic response.[31] The observed negative correlation between IL-9 and HDL-C in the Candida albicans group indicates potential immune-lipid interaction, as HDL-C has known anti-inflammatory properties.[32] These altered relationships underscore the need for species-specific management approaches.[33,34] ROC curve analysis further supports the diagnostic utility of ENO1 and IL-9, with high AUC values indicating strong discriminatory ability.[35] These biomarkers could enhance early detection and monitoring, especially when traditional diagnostics fall short. However, limitations exist. The cross-sectional design precludes causal inference, and the modest sample size may limit generalizability.[36–39] Future longitudinal studies with larger cohorts are needed to confirm these associations and explore mechanisms linking ENO1, IL-9, and Candida infections. Additional research into immune pathways and genetic factors is also recommended.[40,41] In conclusion, ENO1 and IL-9 are closely linked to immune and metabolic changes in Candida infections. Their potential role as diagnostic tools and therapeutic targets merits further investigation.[42,43] Multivariate regression models would better account for the influence of confounding variables, such as BMI, lipid abnormalities, or comorbid inflammation, even though our correlation analyses showed strong associations between ENO1, IL-9, and clinical status. These elements may influence the expression of biomarkers and modify immune responses. One of the study's limitations is the absence of multivariate adjustment. Multivariate linear modeling or logistic regression should be used in future research to separate the impacts of IL-9 and ENO1 on infection status. The dual biomarker approach involving Eno1 and IL-9 offers significant potential for integration into clinical practice, not only for initial diagnosis but also for monitoring treatment response and potentially predicting recurrence in vulvovaginal candidiasis (VC). Elevated Eno1 levels may reflect ongoing fungal activity or incomplete clearance, while persistent or rising IL-9 levels after treatment could indicate sustained immune activation or mucosal vulnerability. Tracking these biomarkers longitudinally could help clinicians evaluate therapeutic effectiveness, allowing for early intervention in cases of inadequate response. Moreover, patients exhibiting a characteristic biomarker pattern, such as elevated baseline IL-9 or recurrent spikes in Eno1, may be at higher risk for relapse, making this profile useful for predictive stratification and tailored follow-up plans. Thus, this biomarker pair could serve as a dynamic tool in both the management and prevention of recurrent VC, improving individualized care.

Despite the high diagnostic performance (AUC 0.883–1.00) of ENO1 and IL-9, practical considerations such as cost, infrastructure, and turnaround time remain important for clinical application, especially in low-resource settings. While ELISA is commonly used in laboratory settings, its routine use for VC diagnosis may not be feasible without simplification or cost-reduction strategies. Current diagnostic methods, like microscopy and culture, are more accessible but have limitations in sensitivity and time efficiency. Therefore, future research should explore point-of-care adaptations, such as lateral flow assays, to make biomarker testing more feasible and cost-effective for widespread use.

To better assess the independent diagnostic value of ENO1 and IL-9, we performed multivariate logistic regression analyses adjusting for BMI and lipid profile. Both biomarkers remained significant predictors, suggesting their association with vaginal candidiasis is not solely confounded by metabolic status. Additionally, effect size metrics such as Cohen's d and adjusted odds ratios were calculated to better contextualize clinical significance. The integration of these biomarkers into routine clinical workflows could thus lead to improved diagnostic precision, better patient outcomes, and more efficient use of healthcare resources. Secretion-based markers (especially ENO1) had the highest effect sizes and odds ratios, indicating their superior diagnostic potential. Serum biomarkers also showed strong associations, though slightly less dramatic. Differences between C. albicans and non-albicans were minimal for IL-9, but moderate for ENO1 secretion, suggesting some discriminatory value.

Limitations

This study has several limitations that should be considered when interpreting the findings. First, its

cross-sectional design prevents establishing causal or temporal relationships between vaginal *Candida* infections and the observed metabolic or immunological changes. Although elevated ENO1 and IL-9 levels were detected, it remains unclear whether these changes are a cause or consequence of infection. Future longitudinal studies are needed to monitor biomarker dynamics over time and assess their predictive or pathogenic roles. Second, the sample size—comprising 120 participants (80 cases and 40 controls)—is modest for biomarker validation and may limit the statistical power, particularly in subgroup analyses. Third, all participants were recruited from a single center (Kamal Al-Samraa Hospital, Baghdad), which may introduce selection bias and limit the generalizability of the findings to broader populations. Factors such as regional, genetic, socioeconomic, and healthcare-related differences could influence both infection dynamics and biomarker expression. Future multi-center studies with larger and more diverse cohorts are recommended to increase statistical robustness and improve external validity. Third, we acknowledge the small sample size as a limitation. Additionally, we propose future multicenter studies with larger, more diverse cohorts to increase the statistical power and generalizability of the results. Fourth, although standard clinical assessments were performed, specific testing for other inflammatory or infectious conditions—such as bacterial vaginosis, sexually transmitted infections, or autoimmune disorders—was not conducted. Therefore, we cannot fully exclude the possibility that elevated ENO1 and IL-9 levels may reflect concurrent or unrelated inflammatory processes. Future studies should incorporate broader diagnostic screening to control for such confounding factors and improve biomarker specificity. Larger and more diverse study populations, along with better control of confounding factors and the use of molecular diagnostics, would strengthen the validity and clinical relevance of future research.

Conclusion

This study provides evidence that Enolase 1 and Interleukin-9 are significantly elevated in both serum and vaginal secretions of patients with vaginal candidiasis, whether caused by *Candida albicans* or non-*Candida albicans* species, in comparison to healthy controls. In the control group, serum ENO-1 showed significant positive correlations with IL-9 in both serum and secretions, as well as with ENO-1 levels in secretions. In the non-*Candida albicans* group, ENO-1 in serum correlated positively with serum IL-9 and negatively with secreted IL-9. These associations suggest that ENO-1 and IL-9 may be involved in the host immune response to fungal infection and could serve as candidate biomarkers reflecting the presence of vaginal candidiasis.

Acknowledgment

We are grateful to the hospital staff for providing the assistance we required to conduct the research.

Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm:
- All the Figures and Tables in the manuscript are ours. Any Figures and images that are not ours have been included with the necessary permission for republication, which is attached to the manuscript.
- No animal studies are present in the manuscript.
- Authors signed off on ethical considerations' approval.
- Ethical Clearance: The project was approved by the local ethical committee at University of Baghdad.

Authors' contributions statement

S.I.A, T.H.M were responsible for the conception and design of the study; S.I.A, T.H.M performed data collection SIA, T.H.M performed data analysis and drafted the article. SIA supervised the study, contributed to data analysis, interpretation, and critical revisions. All authors approved the final manuscript).

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تقييم إينوليز 1 و انترلوكين -9 كمؤشرات حيوية لتحديد شدة داء المبيضات المهبلي في النساء العراقيات

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الخلاصة

داء المبيضات المهبلي، هو عدوى فطرية تسببها فطريات المبيضات البيضاء. هدفت الدراسة إلى استكشاف Eno1 وIL-9 في الفسيولوجيا المرضية لداء المبيضات المهبلي وتحديد ارتباطات القدرة مع معايير القياسات البشرية وملف الدهون. أجريت الدراسة على 120 فردًا، بما في ذلك 80 مريضًا تم تشخيص إصابتهم بداء المبيضات المهبلي و40 سليمًا/ضوابط. تم تصنيف المشاركين إلى مجموعات مصابة بداء المبيضات المهبلي ومجموعات غير مصابة بداء المبيضات المهبلي بناءً على العلامات الطبية والأعراض والنتائج الميكروبيولوجية. تم قياس إفرازات المصل والمهبل من Eno1 وIL-9 باستخدام ELISA، في حين تم قياس ملف الدهون يدويًا باستخدام مطياف ضوئي. لاحظت النتائج زيادة كبيرة في مستويات Eno-1 وIL-9 في كل من مصل وإفرازات المهبل لدى المرضى المصابين بداء المبيضات المهبلي وغير المصابين بداء المبيضات المهبلي مقارنة بعوامل التحكم ($p < 0.001$). تم تحديد ارتباط قوي بين نطاقات IL-9 في المصل و Eno1 في الإفراز لدى المرضى المصابين بـ Candida albicans ($r = 0.440$، $p < 0.01$). أثبت مجموع Eno1 و IL-9 في الإفراز دقة التشخيص المتقدمة للمرضى المصابين بـ Candida albicans (AUC = 1.00، AUC = 0.999) أثناء المصل (AUC = 0.883، AUC = 0.856). بالإضافة إلى ذلك، أدى مجموع Eno1 و IL-9 في الإفراز إلى زيادة دقة التشخيص المتقدمة للمرضى غير المصابين بـ Candida albicans (AUC = 1.00، AUC = 1.00) وكذلك في المصل (AUC = 1.00، AUC = 0.984). تسلط هذه النتائج الضوء على قدرة Eno1 و IL-9 كعلامات حيوية موثوقة لتصنيف شدة داء المبيضات المهبلي، مما يوفر أداة ثمينة لتقنيات العلاج المخصصة والنتائج العلمية المتقدمة لدى النساء.

الكلمات المفتاحية: المبيضات البيضاء، إينوليز-1، إنترلوكين-9، غير المبيضات البيضاء، داء المبيضات المهبلي.