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ALTERED SERUM MARKER ENZYMES IN MALARIA AND DENGUE FEVER – A COMPARISON

Biochemistry

Dr Shiva Vasudev Assistant Professor, Department of Biochemistry, Government Medical College, Kota, Rajasthan

Dr Remesh Kunjunni Assistant Professor, Department of Biochemistry, Government Medical College, Kota, Rajasthan

Dr Nidhi Sharma Senior Demonstrator, Department of Biochemistry, Government Medical College, Kota, Rajasthan

Dr Monika Rathore* Senior Demonstrator, Department of Biochemistry, Government Medical College, Kota, Rajasthan*Corresponding Author

ABSTRACT

Background: Malaria and dengue are significant, seasonally endemic vector-borne diseases in Rajasthan, typically peaking in the post-monsoon period. Malaria is an acute, life-threatening fever, whereas dengue fever causes intense headache and muscle or joint pain. The current study is aimed at learning the changes in the serum enzyme markers in malaria and dengue fevers. **Methods:** The study group included 80 cases (40 malaria and 40 dengue), and 38 healthy controls. We confirmed the malaria and dengue-positive cases using a combination of blood microscopy, rapid diagnostic tests (RDTs), and correlated them with their clinical presentations. Major enzyme markers were investigated and compared with those of healthy subjects. **Results:** We observed a potential elevation of serum aminotransferases (AST and ALT) in both fevers. However, a significant ALT peak was evident in malaria, and a substantial AST elevation was observed in dengue as compared to controls. Serum LDH was also significantly elevated in both fevers, with higher significance in malaria cases rather than in dengue cases. Serum ACP and ALP also showed a similar pattern of rise, indicating potential hepatic involvement. **Conclusions:** Serum enzyme markers are valuable tools in the differential diagnosis of malaria and dengue, particularly for assessing haemolysis and liver damage, as supportive diagnostics when blood smears are inconclusive.

KEYWORDS

Serum enzymes, Malaria, Dengue fever

INTRODUCTION

Alterations in serum enzyme levels are important biochemical indicators in infectious diseases such as malaria and dengue fever, both of which are major public health concerns in tropical and subtropical regions. These infections frequently involve red blood cells, liver and other tissues, leading to measurable changes in circulating enzymes, particularly hepatic transaminases.

Malaria, caused by Plasmodium species, has a well-recognised hepatic phase in its life cycle. During infection, parasitised erythrocytes and inflammatory processes contribute to hepatocellular injury. This results in elevated serum levels of enzymes such as aspartate transaminase (AST), alanine transaminase (ALT), and alkaline phosphatase (ALP). The mechanisms include microvascular obstruction due to infected red blood cells, oxidative stress, and impaired hepatic blood flow, all of which lead to hepatocyte damage and leakage of intracellular enzymes into circulation [1]. The degree of enzyme elevation often correlates with disease severity and may serve as a prognostic marker. Dengue fever, a viral infection caused by the dengue virus, is also commonly associated with hepatic dysfunction. Elevated liver enzymes are among the most frequent laboratory abnormalities observed in dengue patients. Studies have shown that AST and ALT levels are raised in a large proportion of cases, often with AST levels exceeding ALT, which is somewhat distinctive compared to other viral hepatitis [2]. This pattern may be explained by the involvement of not only hepatocytes but also muscle cells, contributing to increased AST levels. The pathogenesis involves direct viral injury to liver cells, immune-mediated damage, and cytokine-induced inflammation [3]. In severe dengue, marked elevations in these enzymes are associated with complications such as haemorrhage, shock, and multi-organ dysfunction [4].

Both malaria and dengue can therefore produce similar biochemical profiles, particularly elevated transaminases, which may complicate differential diagnosis in endemic regions. Importantly, monitoring serum enzyme levels provides valuable insight into the extent of organ involvement, disease progression, and prognosis in both conditions.

In short, altered serum enzymes, especially liver enzymes, are significant markers in malaria and dengue infections. Their evaluation not only reflects underlying pathophysiological processes but also plays a crucial role in clinical assessment and management of these diseases.

MATERIALS AND METHODS

The present study was conducted in the Department of Biochemistry, Sawai Man Singh (SMS) Medical College, Jaipur, Rajasthan, in accordance with the formalities of the institutional ethics committee (IEC). The study group consisted of 40 malaria patients, 40 dengue fever patients, and 38 healthy subjects. Patients and controls in the age group 18 to 50 were recruited according to the pre-designed protocol. Pregnant women, patients with anaemia, liver diseases, haematological disorders and other severe conditions like neurological diseases, cancer and cardiac disorders were excluded.

From each candidate, about 5 mL of venous blood was collected aseptically, and the serum was separated as per the standard protocol. All the symptomatic patients of malaria, confirmed using blood microscopy, rapid diagnostic tests (RDTs), and their clinical presentations, were correlated. The marker enzymes were studied on a fully automated clinical chemistry analyser, AU680 (Beckman Coulter Diagnostics, CA, USA), based on spectrophotometric principles. The samples were run in triplicate, and the mean results obtained are represented as mean ± SD. We have used a nonparametric statistical tool, the Student's t-test, for comparing one variable between two independent samples or groups. Correlation analysis was done by the Spearman rank correlation method. A p-value < 0.05 was considered to be significant, and a p-value of < 0.01 was considered to be highly significant for a given hypothesis testing. All statistical analyses were performed using GraphPad Prism Ver. 6.0 (GraphPad Software, Inc., CA, USA) and Microsoft Excel, MS Office Professional 2021 (Redmond, WA, USA).

RESULTS

In the present study, we recruited 118 candidates, of whom 40 (33.9%) were malaria patients, 40 (33.9%) were dengue fever patients, and 38 (32.2%) were healthy control subjects (Fig. 1A). A significant rise in serum LDH (Fig. 1B) was noted in malaria (291.35 ± 242.61 U/L) and dengue fever cases (183.44 ± 159.26 U/L) as compared to the control group (112.86 ± 109.42 U/L). The rise in LDH was relatively higher in malaria cases than in dengue. The serum CK level (Fig. 1C) and serum GGT level (Fig. 1D) were higher in dengue cases, but not evident in malaria.

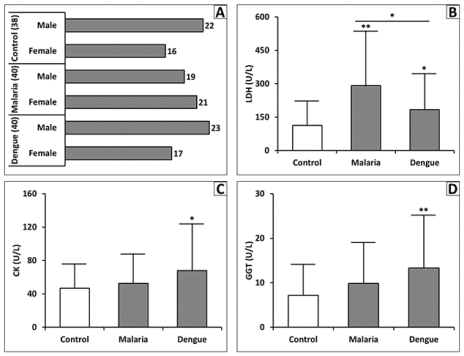

Figure 1: Distribution of cases and controls (A) in the study. Comparison of serum LDH level (B), serum CK level (C), and serum GGT level (D) among the groups. **p-value < 0.01, *p-value < 0.05.

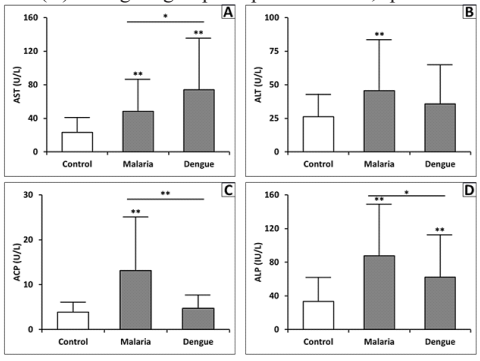

Figure 2: Comparison of serum AST level (A), serum ALT level (B), serum ACP level (C), and serum ALP level (D) among the groups. **p-value <0.01, *p-value <0.05.

We observed a highly significant elevation (p-value: 0.0004) of AST (Fig. 2A) in malaria cases (48.46 ± 37.88 U/L), and a significant rise (p-value: < 0.01) in dengue fever (74.14 ± 61.21 U/L) when compared to that of the control group (23.18 ± 17.77 U/L). The rise in AST in malaria patients was significant compared to that of dengue fever cases (p-value: 0.0269). When we compared the ALT level (Fig. 2B) among the groups, only malaria cases (45.55 ± 37.91) showed a highly significant (p-value: 0.0051) rise compared to that of the control (26.24 ± 16.67 U/L). Dengue fever (35.78 ± 29.12 U/L) did not show any significant ALT rise (p-value: 0.0819). The rise in serum ACP (Fig. 2C) was highly significant (p-value: < 0.01) in malaria, but no significant change was observed in dengue subjects. However, the rise in ACP was higher in malaria cases (p-value: < 0.01). The elevation in serum ALP (Fig. 2D) was highly significant in both malaria (87.53 ± 61.11 IU/L) and dengue cases (62.34 ± 49.93 IU/L) compared with the control group (33.48 ± 28.27 IU/L).

Table 1: Comparison of mean serum levels of different marker enzymes among the control group, malaria patients, and dengue fever cases.

Serum Parameter	Control (N: 38)	Malaria (N: 40)	p-value	Dengue Fever (N: 40)	p-value
LDH (U/L)	112.86 ± 109.42	291.35 ± 242.61	< 0.0001	183.44 ± 159.26	0.0261
CK (U/L)	46.77 ± 28.83	52.54 ± 35.26	0.4327	67.92 ± 55.66	0.0399
GGT (U/L)	7.17 ± 6.99	9.86 ± 9.12	0.1494	13.33 ± 11.85	0.0069
AST (U/L)	23.18 ± 17.77	48.46 ± 37.88	0.0004	74.14 ± 61.21	< 0.0001
ALT (U/L)	26.24 ± 16.67	45.55 ± 37.91	0.0051	35.78 ± 29.12	0.0819
ACP (U/L)	3.89 ± 2.17	13.14 ± 11.88	< 0.0001	4.75 ± 2.91	0.1447
ALP (IU/L)	33.48 ± 28.27	87.53 ± 61.11	< 0.0001	62.34 ± 49.93	0.0026

DISCUSSION

The present study demonstrates significant alterations in serum enzyme levels in patients with malaria and dengue fever compared to healthy controls, reflecting the extent of cellular injury and organ involvement associated with these infections. The observed biochemical changes provide insight into the underlying pathophysiological mechanisms and may have both diagnostic and prognostic significance. The demographic distribution (Fig. 1A) shows a relatively balanced representation of males and females across all study groups, indicating that the observed biochemical variations are unlikely to be influenced by sex-related differences. This aligns with previous epidemiological observations that both malaria and dengue affect both genders with comparable frequency in endemic regions [5].

Table 2: Correlation of different enzyme levels between the control, malaria patients, and dengue patients.

	Control vs Malaria (r)	p-value	Control vs Dengue (r)	p-value	Malaria vs Dengue (r)	p-value
AST	-0.355	0.028	0.050	0.764	0.146	0.369
ALT	0.067	0.692	0.163	0.324	-0.05	0.759
ACP	-0.104	0.542	0.112	0.502	0.002	0.990
ALP	0.203	0.220	-0.07	0.675	-0.032	0.847
GGT	-0.173	0.300	-0.095	0.566	0.243	0.131
LDH	-0.286	0.082	0.079	0.634	-0.053	0.746
CK	0.131	0.437	-0.026	0.874	0.290	0.070

A significant elevation in serum lactate dehydrogenase (LDH) levels was observed in both the malaria and dengue groups (Fig. 1B), with a more pronounced increase in the malaria group. LDH is a non-specific intracellular enzyme released during tissue breakdown and haemolysis. In malaria, the destruction of parasitised erythrocytes, along with hepatic involvement, contributes to markedly elevated LDH levels [6]. Elevated LDH has also been reported as a marker of disease severity in falciparum malaria [7]. In dengue, increased LDH levels reflect tissue hypoxia, endothelial dysfunction, and hepatic injury, although typically less marked than in malaria [8].

Creatine kinase (CK) levels (Fig. 1C) were significantly elevated in both infections, with higher levels observed in dengue patients. This finding suggests greater muscle involvement in dengue, likely due to virus-induced myositis and increased muscle membrane permeability [9]. Myalgia is a hallmark feature of dengue, and elevated CK levels have been correlated with disease severity and muscle damage [10]. Although CK elevation is also seen in malaria, it is generally less pronounced, reflecting comparatively lower muscle involvement.

Gamma-glutamyl transferase (GGT) levels (Fig. 1D) were notably elevated, particularly in dengue cases. The enzyme GGT is a sensitive marker of hepatobiliary dysfunction and oxidative stress. Dengue virus infection is known to cause significant hepatic involvement, ranging from mild transaminase elevation to severe hepatitis [11]. The higher GGT levels observed in dengue may indicate cholestatic injury or oxidative stress-mediated hepatocellular damage [12]. While malaria also affects the liver, the pattern of enzyme elevation suggests relatively lesser cholestatic involvement compared to dengue.

Overall, the differential pattern of enzyme elevation observed in this study marked LDH elevation in malaria and higher CK and GGT levels in dengue—highlights distinct pathophysiological processes in the two diseases. Malaria is predominantly associated with haemolysis and systemic cellular destruction, whereas dengue exhibits greater muscle and hepatic involvement. These findings are consistent with previous studies and reinforce the potential utility of these enzymes as supportive biomarkers in differentiating febrile illnesses in endemic areas [8, 10].

From a clinical perspective, the assessment of serum LDH, CK, and GGT levels may aid in early diagnosis, differentiation, and evaluation of disease severity in patients presenting with acute febrile illness. This is particularly valuable in resource-limited settings where rapid diagnostic tools may not always be available.

These findings reflect the extent of systemic involvement and tissue injury associated with these infections and highlight the potential diagnostic utility of enzyme markers in differentiating febrile illnesses. The comparative analysis reveals that both malaria and dengue are associated with elevated serum enzyme levels, though the pattern and magnitude of elevation differ between the two diseases. Such variations are indicative of distinct underlying pathophysiological mechanisms. Serum lactate dehydrogenase (LDH)

levels were markedly elevated in malaria patients compared to both dengue cases and controls. LDH is a cytosolic enzyme released during cellular damage and haemolysis. In malaria, particularly in Plasmodium falciparum infection, intravascular haemolysis of parasitised erythrocytes is a major contributor to elevated LDH levels. Additionally, hepatic dysfunction and microvascular sequestration further enhance tissue hypoxia and cellular injury, leading to increased LDH release. Previous studies have identified LDH as a useful marker of disease severity and haemolytic burden in malaria [13, 14]. Although dengue also showed elevated LDH levels, the rise was comparatively moderate, suggesting relatively lesser haemolytic involvement.

Creatine kinase (CK) levels were significantly higher in dengue patients than in malaria cases. This observation is consistent with the known myotropic effects of the dengue virus. Muscle involvement, including myositis and increased muscle membrane permeability, is a well-recognised feature of dengue infection and contributes to elevated CK levels (Kalita). The characteristic severe myalgia associated with dengue ("breakbone fever") further supports this finding. In contrast, CK elevation in malaria, although present, is typically less pronounced and may be attributed to generalised systemic inflammation rather than direct muscle injury.

Gamma-glutamyl transferase (GGT) levels were also elevated, with dengue patients demonstrating higher levels compared to malaria patients. GGT is a sensitive marker of hepatobiliary dysfunction and oxidative stress. Dengue infection is frequently associated with hepatic involvement, ranging from mild transaminase elevation to severe hepatitis and, in rare cases, acute liver failure [11]. The increased GGT levels observed in this study may reflect cholestatic changes or oxidative stress-mediated hepatocellular injury. Malaria can also involve the liver; however, the pattern of enzyme elevation suggests comparatively lesser cholestatic injury.

Taken together, the differential enzyme profile observed marked LDH elevation in malaria and higher CK and GGT levels in dengue, underscoring the distinct pathological processes involved. Malaria is primarily characterised by haemolysis and systemic cellular destruction, whereas dengue shows more pronounced muscle and hepatic involvement. These findings are consistent with earlier reports and reinforce the role of biochemical markers in understanding disease mechanisms [8, 11, 14].

From a clinical standpoint, these enzyme alterations may serve as supportive tools in differentiating malaria from dengue in patients presenting with acute febrile illness, particularly in endemic regions where co-circulation of both diseases is common. In resource-limited settings, where access to advanced diagnostics may be restricted, such biochemical parameters can provide valuable adjunctive information. The findings of our study are comparable to other recent prospective studies [15].

However, the study has certain limitations. The sample size may limit the generalizability of the findings, and the cross-sectional design does not allow assessment of temporal changes in enzyme levels during the course of illness. Future studies incorporating larger cohorts, longitudinal follow-up, and correlation with disease severity are warranted to validate and extend these findings.

CONCLUSIONS

The present study demonstrates significant alterations in serum enzyme profiles in patients with malaria and dengue fever compared to healthy controls, reflecting varying degrees of tissue injury and organ involvement. A distinct pattern of enzyme elevation was observed, with markedly increased LDH levels in malaria, indicative of haemolysis and systemic cellular damage, while dengue patients exhibited higher CK and GGT levels, suggesting greater muscle involvement and hepatobiliary dysfunction. These differences highlight the distinct pathophysiological mechanisms underlying the two infections. From a clinical perspective, serum enzymes such as LDH, CK, and GGT may serve as useful adjunct biomarkers in differentiating malaria from dengue, particularly in endemic and resource-limited settings. Additionally, their levels may provide insight into disease severity and the extent of organ involvement.

However, limitations such as small sample size and lack of longitudinal follow-up restrict the generalizability of the findings. Further large-scale, prospective studies are warranted to validate these observations and explore their prognostic significance.

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CONFLICT OF INTERESTS

The authors hereby declare that they have no conflicts of interest related to this original work.

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