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Research Article

A New Perspective on Dengue Severity: Elevated Serum Amyloid A and its Clinical Significance in Hemorrhagic Fever

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I N F O

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A B S T R A C T

Background: Dengue fever, caused by four distinct RNA virus serotypes of the flaviviridae family, has become increasingly prevalent worldwide. Serum amyloid A has been implicated in various inflammatory diseases, but its role in dengue is not thoroughly explored. Our study was aimed at evaluating the serum levels of SAA in individuals at various stages of dengue severity.

Methods: The cross-sectional study recruited 20 healthy participants aged 18-30 years from a tertiary care hospital in Punjab, i.e., DHF patients (n=20), and 20 healthy participants from outside the hospital. Biochemical assessments included measurements of TNF-α, IL-6, IL-13 and SAA using ELISA kits.

Results: DHF patients exhibited elevated levels of SAA, AST, ALT, IL-6, IL-13, TNF-α and CRP compared to healthy control ($p < 0.0001$). SAA levels correlated negatively with platelet count (r = -0.9694) and positively with haematocrit (r = 0.8996), TNF-α (r = 0.8273), CRP (r = 0.8368), IL-6 (r = 0.6154), IL-13 (r = 0.8397), AST (r = 0.7720), and ALT (r = 0.7363) in DHF patients.

Conclusion: Elevated SAA levels were found to be strongly correlated with the severity of DHF, correlating with various haematological, biochemical, and inflammatory markers. SAA may serve as a valuable diagnostic biomarker for evaluating the disease severity in dengue patients.

Keywords: Serum Amyloid A, Dengue, Inflammation, Dengue Haemorrhagic Fever, Interleukins

Introduction

One of the four distinct RNA virus serotypes that cause dengue fever (DF), which are members of the Flaviviridae. DF is mostly spread by female Aedes aegypti mosquitoes.[1] Dengue incidence reached a record high in 2023, impacting more than 80 nations. Since the outbreak of 2023, continuous transmission, along with an unanticipated rise in dengue infections, has led to an all-time record of over 6.5 million reported illnesses and over 7300 dengue-related fatalities.[2]

The clinical presentation of dengue can be asymptomatic or symptomatic depending on the case. Mild symptoms to severe dengue haemorrhagic fever (DHF) are among the symptoms of DF[3]. The WHO 2009 recommendations, which describe DHF as an infection with dengue with indicators such as plasma leakage and thrombocytopenia, along with bleeding symptoms, served as the basis for the criteria used to identify patients.[4] DHF is marked by signs such as bleeding that occurs spontaneously, a considerable drop in platelet cells and increased vascular permeability seen as enhanced haemoconcentration, pleural effusion or ascites.[5] Severe dengue is accompanied by a transitory increased blood vessel permeability and plasma leakage owing to endothelial cell failure.[6,7] Endothelial cells produce a selective physical barrier that allows molecules to exchange between the cells and lining capillaries and blood vessels. It has been shown that the NS1 protein directly contributes to vascular leakage by breaking down the glycocalyx lining the endothelium's sialic acid and heparan sulphate proteoglycans.[8,9] Previous research has shown that hospitalised dengue patients had elevated levels of several cytokines, chemokines, and lipid markers.[10] Certain DENV proteins cause inflammation by stimulating immune cells and producing cytokines and chemokines that promote inflammation. Through the NF-κB pathway, the E protein domain III region initiates the synthesis of cytokines.[11]

One of the most significant components of the immune response is serum amyloid A (SAA).[12] The pathophysiological implications of SAA have been extensively researched over six decades. Understanding its function and regulation is essential for elucidating its role in various disease processes.[13] SAA is an acute-phase protein primarily synthesised in the liver in response to inflammatory cytokines such as IL-1β, IL-6, and TNF-α. While in the intestines, SAA affects epithelial barriers, encourages the development of T cells, and makes it easier to engulf Gram-negative bacteria, all of which contribute to inflammatory bowel disease.[14] In emergency situations like inflammation, trauma, and viral infection, the body's SAA levels may rise quickly - up to a thousand times.[14] SAA stimulates chemotaxis and increases calcium flow by binding to FPLR1 and FPLR2 in HEK293 cells and HUVECs[15]. According to several investigations, plasma SAA levels outperformed the commonly used clinical biomarker of inflammation, CRP, as a predictor of future cardiovascular events.[16,17,18,19,20,21] It was recently claimed that SAA contributes to atherosclerosis and associated consequences.[22] The earlier research suggests that increased SAA levels throughout a variety of inflammatory diseases may be related to the severity of the illness.[23] Clinical indicators such as blood platelet counts, haematocrit levels, and indications of plasma leakage were used to evaluate severity in addition to serological results. Therefore, the current study aimed to evaluate the SAA levels in DHF patients and their association with inflammatory biomarkers influenced by illness severity.

Material and Methods

Study participants

The research participants aged 18 to 30 years were recruited from the tertiary care hospital, Punjab, in 2023-2024, following permission by the institutional ethical committee and healthy volunteers outside the hospital premises. ELISA kits for the detection of dengue IgM antibodies were used for confirming dengue infection in recruited patients. Patients with detectable IgM were categorised as having infections. The participants were categorised into two types: healthy control and DHF, having 20 participants in each group. Patients with cancer, haematological problems, or heart issues were excluded from the research. All participants were told about the trial, and each participant signed an informed consent form prior to being included. The study was conducted as per the Declaration of Helsinki (DoH), as amended when necessary, and ICH E6-GCP standards.

Biochemical Assessment

A total of ten millilitres of venous blood samples were collected from each of the participants. The serum was extracted from the whole blood by centrifugation, which was run for 15 minutes at 1300 rpm. We purchased ELISA kits for TNF-α, IL-13 and SAA from Progenbiolab Technologies Pvt. Ltd, Delhi, India.

Statistical Analysis

All the normally distributed and non-parametric variables were plotted using the arithmetic mean and SD. The significance between the study groups was indicated using unpaired Student's t-test. The univariate analysis was assessed using Spearman's correlation (p value < 0.05). All of the data analysis was done using GraphPad Prism 9, a statistical programme.

Results

Clinical Characteristics

Table 1 shows the baseline biochemical and demographic features of the 20 DHF and 20 healthy participants (control group). The dengue-infected population was mostly DENV-2 seropositive compared to DENV-3 seropositive, according to the RT-PCR data. The investigation focuses on indicators of illness severity at later phases, where DENV IgM screening is more pertinent. 'NS1 antigen positive' is commonly used for early-stage dengue identification (during the first 5-7 days after symptom onset), although IgM antibodies are detected later. Because the research sample comprised individuals with advanced illness (DHF), IgM testing was preferred to NS1 antigen testing.

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Comparison of healthy control subjects and DHF group

When compared to the control group, individuals with DHF had statistically substantially fewer platelets (Table 1). Subjects with DHF had substantially higher haematocrit levels than the control group (Table 1). CRP levels between DHF patients and control participants were significantly different (Table 1). DHF patients had higher levels of AST, ALT and TNF-α.

Spearman Correlation of SAA with Inflammatory Markers and Clinical Variables in the DHF Group

Data showed significant negative correlation with platelets (r= -0.9694) and positive correlation with haematocrit (r = 0.8996), TNF-α (r = 0.8273), CRP (r = 0.8368), IL-13 (r = 0.8397), and AST (r = 0.7720) with $p < 0.0001$, respectively; IL-6 (r = 0.6154, $p = 0.0039$); and ALT (r = 0.7363, $p = 0.0002$) (Figure 1).

Table 1.Demographic and physiological laboratory parameters of the study groups (healthy control and DHF)

Variables	Healthy Control	DHF	*p* value
n	20	20	-
Age	25.3 ± 4.21	23.5 ± 3.76	-
Platelets (cells/mm^3)	301070.3 ± 75260.88	68138.1 ± 6870.53	*<0.0001*
Hematocrit (%)	32.3 ± 3.45	43.9 ± 6.2	*<0.0001*
AST (IU/L)	37.1 ± 3.13	88.2 ± 16.88	*<0.0001*
ALT (IU/L)	51.7 ± 5.37	72.11 ± 7.85	*<0.0001*
CRP (mg/L)	0.9 ± 0.11	10.54 ± 3.28	*<0.0001*
TNF-α (pg/ml)	28.53 ± 2.50	150.23 ± 16.15	*<0.0001*
IL-6 (pg/ml)	5.34 ± 1.72	96.825 ± 16.75	*<0.0001*
IL-13 (pg/ml)	4.67 ± 1.84	116.45 ± 25.81	*<0.0001*
SAAP (µg/ml)	11.48 ± 3.42	107.45 ± 28.55	*<0.0001*

Table 2.The univariate analysis between SAA levels and biochemical parameters of DHF subjects

Variables	Univariate Analysis	
	R	***p***
SAA		
Platelets	-0.9694	*<0.0001*
Hematocrit	0.8996	*<0.0001*
TNF-α	0.8273	*<0.0001*
CRP	0.8368	*<0.0001*
IL-6	0.6154	*0.0039*
IL-13	0.8397	*<0.0001*
ALT	0.7363	*0.0002*
AST	0.7720	*<0.0001*

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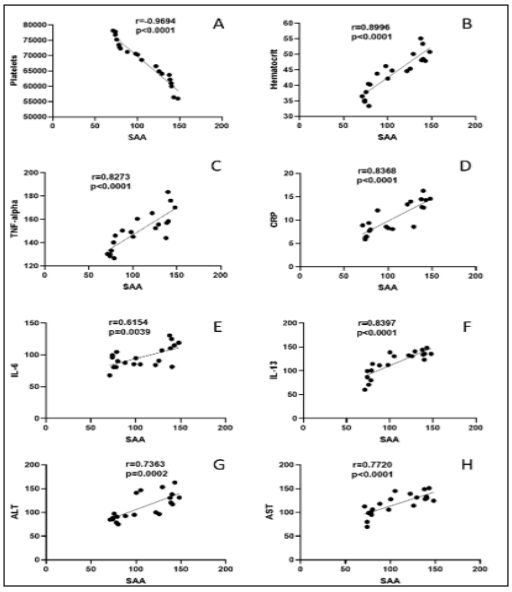

Figure 1.Spearman correlation among SAA, platelets, hematocrit, TNF-α, CRP and inflammatory markers in DHF patients and liver enzymes. The correlation is depicted between SAA and platelets values in panel (A), SAA and hematocrit levels in panel (B).

Discussion

Dengue is a dynamic, complicated, and fast-moving illness that is caused by complex interactions between the virus, host, and vector.[24] DF often advances quickly to its severe form, DHF. DHF is the leading cause of death.[25,26] However, effective illness management significantly improves the prognosis of patients. Severe dengue illness progression is linked to a number of parameters, including host immunological and genetic factors, virus load, serotype, and virulence.[27,28] Numerous investigations have assessed the clinical factors' ability in predicting the progression of severe dengue.[29,30] Early dengue infection is characterised by high levels of inflammatory markers. These mediators are further linked to a number of sequelae, including shock, haemorrhage, and vascular leakage.[28] There is evidence to suggest that dengue severity is greatly impacted by altered coagulation profiles, elevated HCT, and reduced platelet counts. The DHF/DSS (dengue shock syndrome) condition was linked to a significant fall in platelet count (<20,000/mm^3) in those who had mild to moderate thrombocytopenia, according to an experimental trial including 105 juvenile dengue-affected patients.[31] Being a sensitive indicator of inflammation, the CRP may be able to assess the severity of DF. In research with 191 dengue-infected participants, DSS group CRP levels were significantly higher than those of DHF and DF groups.[32] Moreover, compared to non-severe dengue, Atakuri et al.'s study also found a significant connection (p<0.0001) between dengue severity and CRP level.[33] Comparatively similar results were observed in our study, as CRP levels were higher in the DHF group, as DHF represents a severe form of dengue, characterised by extensive inflammation. The body's immune response to the dengue virus includes the activation of various inflammatory pathways, leading to increased CRP production.

In this study, TNF-α levels were observed to be elevated in DHF patients. This could lead to plasma leakage, haemorrhage, and shock. According to Kumar et al., the DHF had statistically higher TNF-α levels. This suggests that the TNF-α level may be used as a predictor of dengue infection severity and plays a major role in DHF.[34] These studies concluded that raised TNF-α production plays a role in DHF by functioning at the vascular endothelium. Masyeni et al. show a higher level of IL-6 in dengue patients was strongly correlated with nausea/vomiting and bleeding, though the strength of the correlation coefficients varied.[37] The study conducted cytokine profiling during a dengue epidemic in Taiwan and found that DHF patients had elevated levels of IL-13[38]. Similarly, both the cytokines i.e. IL-6 and 13, were found to be elevated in DHF patients. High IL-6 levels in DHF may indicate severe inflammation and vascular permeability. Elevated IL-13 levels reflect skewed Th2 immune responses, contributing to immune dysregulation and exacerbating disease pathology.

Shah et al. reported the serum AST and ALT levels were markedly raised in DHF/DSS patients, and they also found a correlation with the infection's severity.[39] In the present study, AST and ALT levels were greater in DHF. It is believed that a combination of direct viral impacts and the host's immunological response, which causes inflammation and cell destruction, causes liver damage. Indicators indicating the severity of the illness, such as elevated liver enzymes

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in DHF patients, might aid in the clinical evaluation and treatment of these individuals.

To the best of our knowledge, this is the first study reporting elevated SAA levels in DHF patients and their significant association with liver enzymes and inflammatory cytokines. Previous research has examined SAA levels in various inflammatory diseases.[40] Our findings suggest that SAA could be used as a diagnostic indicator for assessing disease severity in dengue patients.

Conclusion

This work is the first to demonstrate that SAA levels rise noticeably when dengue complications, especially DHF, occur. The results show that a number of haematological, biochemical, and liver-related problems seen in DHF patients are strongly correlated with higher SAA levels. According to these findings, elevated SAA levels may be a useful anti-inflammatory diagnostic marker in dengue patients, especially in those with severe symptoms like DHF. A biomarker like this might help with early disease severity identification and classification, allowing for prompt clinical intervention and improved patient outcomes. Although these findings are encouraging, they must be confirmed in bigger and more varied dengue patient groups because they are based on a small cohort. In order to prove that SAA is a diagnostic tool that can be used anywhere, future research should try to validate these findings across a range of demographic groupings and geographic areas.

Author's Contribution: Conceptualisation: PS & CP; Methodology: PS, RS, RS and SG; Software: CP; Validation: CP, PK, RK and RK; Formal Analysis: CP and RK; Investigation: PS, CP, SG, RS, and RS; Data Curation: CP and SJ; Writing-original draft preparation: CP, PS, PK, SJ and TGS; Writing-review and editing: TGS, SJ, PK and RS.

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