



International Journal of Medicine

<https://www.theinternationalmedicine.com>



Research Article

Clinical features and outcomes of Thrombotic Microangiopathy in Snake Bite

Anjali Raj^{1*}, VIKAS L^{2*}, ³Riyaz Ahmed

¹Resident, Department of General Medicine Mysore Medical College and Research Institute, Mysore, India

²Associate Professor, Department of General Medicine Mysore Medical College and Research Institute, Mysore, India

³Assistant Professor, Department of General Medicine Mysore Medical College and Research Institute, Mysore, India

Received -26/08/2025 | Reviewed - 03/09/2025 | Accepted - 13/09/2025 | Published - 25/09/2025

Abstract

Background: Snakebite envenoming is a significant but under-recognized global health problem, particularly in the Indian subcontinent, where it causes substantial mortality and morbidity. Objective: To assess the clinical features and outcomes of TMA in snakebite patients at a tertiary care hospital in India. **Methods:** The study population comprised patients admitted following snakebite. Data was collected using a pre-structured proforma and included demographics, clinical features, and serial laboratory investigations (WBCT, PT/INR, complete hemogram, LFT, RFT). TMA was defined as microangiopathic hemolytic anemia (MAHA) with $\geq 1.0\%$ schistocytes and thrombocytopenia. Statistical analysis involved descriptive statistics and comparative tests. **Results:** The study included 100 patients. The mean age of patients was 48.29 years, and 56% were male. Common clinical features included local signs of envenomation (79%), coagulopathy (73%), oliguria/hematuria (26%), and jaundice (9%). The overall incidence of TMA was 9%. TMA was significantly associated with oliguria/hematuria and jaundice ($p < 0.001$). TMA patients had significantly deranged hematological and renal parameters. AKI developed in 39% of patients, and TMA was strongly associated with AKI ($p < 0.001$) and increased need for dialysis ($p = 0.004$). **Conclusion:** TMA is a clinically significant complication of snakebite, strongly associated with AKI and adverse hematological and renal profiles. Early recognition of TMA is crucial for prompt management and improving outcomes in snakebite patients.

Keywords: Snakebite, Thrombotic Microangiopathy, Acute Kidney Injury

INTRODUCTION

Snakebite envenoming represents a serious global public health problem, especially in tropical and subtropical areas, yet its true scale is often underestimated. According to the World Health Organization (WHO), up to 5.4 million snakebites may occur annually, leading to as many as 2.7 million cases where venom causes harmful effects (envenoming)¹. The resulting death toll is substantial, estimated at 81,000 to 138,000 fatalities per year². Furthermore, long-term suffering is significant, with approximately 400,000 individuals annually facing permanent disabilities like amputations or chronic conditions after a bite³. Because snakebite disproportionately harms impoverished rural populations, the WHO designates it a high-priority neglected tropical disease (NTD)⁴. Although snakebite envenoming occurs worldwide, its burden is heavily concentrated geographically, with India facing a disproportionately high impact. The landmark Million Death Study estimated that India witnessed approximately 1.2 million snakebite-related deaths from 2000 to 2019, averaging nearly 58,000 fatalities annually⁵. This represents a significant fraction of total global snakebite mortality. A large number of severe envenomings within India are caused by the 'Big Four' snakes: spectacled cobra (*Naja naja*), common krait (*Bungarus caeruleus*), Russell's viper (*Daboia russelii*), and saw-scaled viper (*Echis carinatus*)⁶. Among these, Russell's viper is frequently associated with severe systemic complications like coagulopathy, hemorrhage, and acute kidney injury; the kidney damage, in particular, is often linked to underlying thrombotic microangiopathic (TMA) mechanisms⁷. This extensive mortality and morbidity firmly position snakebite envenoming as a major public health priority requiring dedicated strategies in India⁸. Addressing the knowledge gap on snakebite-associated TMA (SB-TMA) in India is crucial. This study therefore systematically investigates SB-TMA at an Indian tertiary hospital. We aim to determine its local incidence, typical clinical and laboratory features (including MAHA markers, renal function), and key outcomes like acute kidney injury (AKI) severity among admitted snakebite patients. Generating this vital regional data should improve early recognition and risk assessment of SB-TMA in clinical practice. Ultimately, this knowledge is expected to guide better management strategies, potentially including decisions on interventions like dialysis, helping to reduce the significant illness and death caused by severe snakebite envenoming across India.

Address for Correspondence Dr. Anjali Raj, Resident, Department of General Medicine Mysore Medical College and Research Institute, Mysore, India, Email ID: vanjalirajabr@gmail.com

DOI: 10.5455/im.5894121

This is an Open Access article under the terms of the Creative Commons Attribution-Noncommercial 4.0 International License (<https://creativecommons.org/licenses/by-nc/4.0/>)

MATERIALS & METHODS

This Prospective observational study was conducted among patients admitted to Department of General Medicine at Mysore Medical College and Research Institute (MMCRI), Mysore. The study period was 18 months, commencing from the date of obtaining ethical

Inclusion Criteria:

Patients aged >18 years admitted with a history of snake bite in the Department of General Medicine, Government Medical College, Mysore, were included in the study.

Exclusion Criteria:

- Patients with diagnosed microangiopathy were excluded.
- Patients with diagnosed Systemic Lupus Erythematosus (SLE), Scleroderma, or Antiphospholipid Antibody (APLA) syndrome were excluded.
- Patients taking medications known to cause Thrombotic Microangiopathy (TMA), such as Mitomycin, quinine, cyclosporine, tacrolimus, muromonab, interferon, ticlopidine, and clopidogrel, were excluded.

Sample Size Calculation

The sample size was calculated to be 100, using the formula: $S = (4 \times P \times Q) / D^2$

Where:

- S = Required sample size
- P = Estimated prevalence of the condition of interest (thrombotic microangiopathy - TMA)
- 1-P = Estimated proportion of the population without the condition
- D = Desired margin of error

The estimated prevalence (P) of TMA following snakebite can vary considerably across different geographical regions and depending on the snake species involved. As highlighted in the systematic review by Noutsos et al. (2020) 9, the prevalence of TMA in snakebite victims ranges from as low as 5-10% in cases involving various species to higher rates reported in other regions and with different snake families.

The desired margin of error (D) was set at 0.05 (or 5%).

Substituting these values into the sample size formula:

$$S = (4 \times 0.06 \times (1-0.06)) / (0.05^2)$$

$$S = 91$$

Therefore, based on a conservative estimated prevalence of 6% and a desired margin of error of 5%, the minimum required sample size for this study was calculated to be approximately 91 patients.

The present study prospectively enrolled 100 patients admitted. This achieved sample size of 100 exceeds the calculated minimum requirement of 91, ensuring adequate statistical power and precision to estimate the prevalence of TMA and to analyse the secondary outcomes of interest within this study population.

Data Collection

After obtaining approval and ethical clearance from the ethical committee, informed written consent was taken from each participant. Detailed history was recorded, and the following investigations were conducted:

- Whole blood clotting time (WBCT)
- Prothrombin time (PT) / International Normalized Ratio (INR)
- Complete hemogram with peripheral smear for schistocytes and reticulocyte count
- Liver function tests (LFT)
- Renal function tests (RFT)

These investigations were repeated at two hours, six hours, 12 hours, and then 24-hourly until the patient was discharged from the hospital. A pre-structured proforma was used for data collection.

Statistical Analysis: To compare categorical variables, the Chi-square test or Fisher's exact test (where appropriate) was used. To compare means of continuous variables between groups, the independent samples t-test was used if the data was normally distributed and had equal variances. The significance level (alpha) was set at 0.05 for all statistical tests. All statistical analyses were performed using statistical software package (SPSS version 26.0)..

RESULT

The majority of patients (41%) were in the 51–70-year age group, with 41 individuals. The 31–50-year age group comprised 37% of the patients, with 37 individuals. The youngest age group (≤ 30 years) included 15% of the patients (15 individuals), while the oldest age group (≥ 71 years) had the smallest representation, accounting for 7% of the total, with 7 individuals. The mean age of the patients was noted as 48.29 with a SD of 14.25 years.

The study population consisted of 56 male patients, accounting for 56% of the total, and 44 female patients, making up 44% of the total. Among the 15 patients in the ≤ 30 age group, 73.3% were male (11 patients) and 26.7% were female (4 patients). In the 31-50 age group, there were 17 males (45.9%) and 20 females (54.1%), totaling 37 patients. The 51-70 age group comprised 25 males (61.0%) and 16 females (39.0%), with a total of 41 patients. Finally, the ≥ 71 age group included 3 males (42.9%) and 4 females (57.1%), totaling 7 patients. Overall, there were 56 male patients (56.0%) and 44 female patients (44.0%) in the study. The Chi-square test revealed no statistically significant association between age group and gender distribution (Chi-square value = 4.250, $p = 0.236$).

Table 1 – Distribution of patients according to the type of snake bite

S No	Snake	No of Patients	Percentage
1	Viper	33	33.0
2	Unknown	67	67.0
Total		100	100

33 patients (33.0%) were identified as having been bitten by a Viper, while the majority, 67 patients (67.0%), had snake bites of an unknown type.

The majority of patients, 70 individuals (70.0%), presented between 4 to 8 hours after the snake bite. A smaller portion, 27 patients (27.0%), presented in less than 4 hours. Only 3 patients (3.0%) presented more than 8 hours after the snake bite. The mean time to presentation was noted as 6.35 hours with a SD of 4.53 hours.

Table 2 – Distribution of patients based on the clinical features

S No	Local signs of envenomation	No of Patients	Percentage
1	Local signs of envenomation	79	79.0
2	WBCT not clotted	73	73.0
3	Oliguria/Haematuria	26	26.0
4	Jaundice	9	9.0
5	Signs of neurotoxicity	13	13.0

Local signs of envenomation were observed in 79 patients, accounting for 79.0% of the study population. A whole blood clotting time (WBCT) that was not clotted was noted in 73 patients (73.0%). A WBCT that is not clotted suggests that the blood is taking longer than normal to clot, which can be a sign of venom-induced consumption coagulopathy (VICC) - a common complication in snake bites. Oliguria or haematuria was present in 26 patients, representing 26.0%. Jaundice was recorded in 9 patients, which is 9.0% of the total. Signs of neurotoxicity were seen in 13 patients, making up 13.0% of the patients.

Table 3 – Descriptive Statistics of Hematological Parameters in the Study Population

Variable	N	Minimum	Maximum	Median	Mean	Std. Deviation
PT	100	11.00	35.00	18.60	19.36	6.47
APTT	100	18	100	44.50	47.11	18.03
INR	100	.6	5.7	2.35	2.329	1.28
Hb	100	8	15	12.0	12.14	1.54
WBC	100	4200	21000	7450.0	8720.92	3823.41
Platelets	100	35000	415000	189500.0	186280.56	93328.737

Prothrombin time (PT) values ranged from 11.00 to 35.00, with a median of 18.60 and a mean of 19.36 (SD = 6.47). Activated partial thromboplastin time (APTT) values varied from 18 to 100, with a median of 44.50 and a mean of 47.11 (SD = 18.03). The international normalized ratio (INR) ranged from 0.6 to 5.7, with a median of 2.35 and a mean of 2.329 (SD = 1.28). Hemoglobin (Hb) levels ranged from 8 to 15, with a median of 12.0 and a mean of 12.14 (SD = 1.54). White blood cell (WBC) counts ranged from 4200 to 21000, with a median of 7450.0 and a mean of 8720.92 (SD = 3823.41). Platelet counts ranged from 35000 to 415000, with a median of 189500.0 and a mean of 186280.56 (SD = 93328.737).

Table 4 – Descriptive Statistics of Renal Parameters in the Study Population

Variable	N	Minimum	Maximum	Median	Mean	Std. Deviation
Urea	100	7	240	35.0	76.09	64.97
Creatinine	100	.60	11.00	1.22	2.79	2.81
Sodium	100	121	145	136.0	135.25	5.72
Potassium	100	3.1	7.0	4.30	4.767	1.0854

Urea levels ranged from 7 to 240, with a median of 35.0 and a mean of 76.09 (SD = 64.97). Creatinine levels ranged from 0.60 to 11.00, with a median of 1.22 and a mean of 2.79 (SD = 2.81). Sodium levels ranged from 121 to 145, with a median of 136.0 and a mean of 135.25 (SD = 5.72). Potassium levels ranged from 3.1 to 7.0, with a median of 4.30 and a mean of 4.767 (SD = 1.0854). All parameters were measured in 100 patients.

Table 5 – Descriptive Statistics of Liver Function Parameters in the Study Population

Variable	N	Minimum	Maximum	Median	Mean	Std. Deviation
Total Bilirubin	100	0.2	3.5	0.80	.957	0.667
Direct Bilirubin	100	0.1	2.1	0.20	.407	0.493
AST	100	12	262	32.0	46.11	42.02
ALT	100	12	200	35.0	44.42	31.14
LDH	37	110	1982	170	484.38	602.13

Total bilirubin levels ranged from 0.2 to 3.5, with a median of 0.80 and a mean of 0.957 (SD = 0.6676). Direct bilirubin levels ranged from 0.1 to 2.1, with a median of 0.20 and a mean of 0.407 (SD = 0.4926). Aspartate transaminase (AST) values ranged from 12 to 262, with a median of 32.0 and a mean of 46.11 (SD = 42.021). Alanine transaminase (ALT) values ranged from 12 to 200, with a median of 35.0 and a mean of 44.42 (SD = 31.143). Lactate dehydrogenase (LDH) values, measured in 37 patients, ranged from 110 to 1982, with a median of 170 and a mean of 484.38 (SD = 602.132).

Table 6 – Distribution of patients according to ASV administration

S No	ASV	No of Patients	Percentage
1	Administered	92	92.0
2	Not administered	8	8.0
Total		100	100

The majority of patients, 92 out of 100 (92.0%), were administered ASV. A smaller group of 8 patients (8.0%) did not receive ASV. In this study, 39 patients (39.0%) developed AKI, while the majority, 61 patients (61.0%), did not develop AKI. Among the 39 patients who developed AKI, a majority of 23 patients (59.0%) required dialysis, while 16 patients (41.0%) did not require dialysis.

All 9 patients (100.0%) with suspected TMA had schistocytes present.

Among the 9 patients whom renal biopsy was indicated, 8 patients (88.9%) showed fibrinoid necrosis or intraglomerular thrombi deposition on biopsy. Only 1 patient (11.1%) did not undergo biopsy because of high degree of thrombocytopenia.

Table 7 – Distribution of patients on the basis of development of features of TMA

S No	Features of TMA	No of Patients	Percentage
1	Developed	9	9.0
2	Not developed	91	91.0
Total		100	100

In this study, 9 patients (9.0%) developed features of TMA, while the majority, 91 patients (91.0%), did not develop TMA. The association between demographic and snakebite characteristics with the presence of thrombotic microangiopathy (TMA).

- Age Group: The distribution of TMA across age groups was as follows: Age group ≤30 years) had 11.1% of TMA cases, age group of 31-50 years had 44.4%, age group of 51-70 years also had 44.4%, and age group of ≥71 years had no TMA cases (0.0%).
- Gender: TMA was present in 33.3% of males and 66.7% of females.
- Snake Type: 55.6% of Viper bites resulted in TMA, while 44.4% of Unknown snake bites led to TMA.
- Time of Presentation: TMA occurred in 11.1% of patients presenting <4 hours, 77.8% of patients presenting between 4-8 hours, and 11.1% of patients presenting >8 hours.

However, the Chi-square tests indicated that none of these associations (Age group, Gender, Snake type, Time of presentation) were statistically significant with the presence of TMA ($p > 0.05$ for all tests).

The presence of oliguria/hematuria and jaundice showed a strong and statistically significant association with the presence of TMA in this study.

Parameters PT, INR, Hb, and Platelets showed statistically significant differences between the two groups.

Discussion:

This finding of a higher prevalence of snakebites in the middle to older age groups aligns with observations in other studies that have reported on the age demographics of snakebite victims. For instance, Ihsan et al. (2022) 10 also noted a substantial representation of middle-aged individuals in their snakebite cohort. Similarly, Sasidharan et al. (2025) 11 highlighted age as a significant factor in snakebite epidemiology. The gender distribution in this study revealed a slightly higher proportion of male patients (56%) compared to female patients (44%). This

observation is consistent with several other studies that have reported a male preponderance in snakebite cases. Chandrakumar et al. (2016) 12 reported a male-to-female ratio of 1.93 in their study, indicating a higher incidence of snakebites among males.

The combined analysis of age group and gender in this study did not reveal a statistically significant association (Chi-square $p = 0.236$). While there were variations in the gender distribution within each age group, these differences were not significant enough to establish a relationship between age and gender in snakebite incidence. This finding contrasts somewhat with studies like Chandrakumar et al. (2016) 12 and Sasidharan et al. (2025) 11, which have explored the interplay of age and gender in the context of occupational and behavioral risk factors. In this study, a significant proportion (67%) of snakebites were attributed to "Unknown" snake types, while 33% were identified as Viper bites. The high percentage of unidentified snake types is a common challenge in snakebite epidemiology studies, as accurate identification can be difficult due to various factors, including the patient's inability to identify the snake, nocturnal bites, and rapid disappearance of the snake. Chandrakumar et al. (2016) 12 also reported a substantial proportion (52.7%) of unidentified snake species in their study.

The time to presentation in this study ranged from less than 4 hours to more than 8 hours, with a mean time of 6.35 hours (SD = 4.53 hours). The majority of patients (70%) presented between 4 and 8 hours after the snakebite, while 27% presented in less than 4 hours, and only 3% presented after 8 hours. The importance of early presentation in snakebite management has been emphasized in several studies. Sujir et al. (2018) 13 found that patients who arrived at the hospital within 6 hours of the snakebite required less ASV and experienced fewer complications.

The clinical features observed in this study included local signs of envenomation (79%), a WBCT that was not clotted (73%), oliguria/hematuria (26%), jaundice (9%), and signs of neurotoxicity (13%). Local signs of envenomation are the most common presentation in snakebite victims. The finding of a WBCT that was not clotted in a substantial proportion of patients (73%) indicates the presence of coagulopathy, which is a frequent complication of snakebite envenomation, particularly following Viperidae bites.

Alvitigala et al. (2024) 14 provided a detailed review of the mechanisms by which snake venoms disrupt hemostasis, leading to various clinical syndromes, including venom-induced consumption coagulopathy (VICC). Oliguria/hematuria, observed in 26% of patients, is a significant indicator of renal involvement and the potential development of AKI, a major complication of snakebite.

The hematological parameters in this study revealed a range of values, with mean PT, APTT, INR, Hb, WBC, and platelet counts of 19.36 (SD = 6.47), 47.11 (SD = 18.03), 2.329 (SD = 1.28), 12.14 (SD = 1.54), 8720.92 (SD = 3823.41), and 186280.56 (SD = 93328.737), respectively. These values indicate that snakebite envenomation can lead to significant hematological abnormalities. Specifically, the alterations in PT, APTT, and INR suggest coagulopathy, a common complication of snakebites. The reduced platelet count is particularly relevant in the context of TMA, as thrombocytopenia is a key diagnostic criterion. Studies such as Isbister et al. (2007) 15, Noutsos et al. (2022) 16 have emphasized the importance of thrombocytopenia, along with microangiopathic hemolytic anemia (MAHA), in defining snakebite-associated TMA. Anemia, as reflected in the Hb levels, is also a significant finding, which can be attributed to hemolysis or bleeding.

Renal Parameters

The renal parameters in this study showed a mean urea level of 76.09 (SD = 64.97) and a mean creatinine level of 2.79 (SD = 2.81), indicating varying degrees of renal dysfunction. Sodium levels had a mean of 135.25 (SD = 5.72), and potassium levels had a mean of 4.767 (SD = 1.0854). These findings are consistent with previous studies that have documented the impact of snakebite envenomation on renal function. Vikrant et al. (2017) 17 and Rao et al. (2019) 18 extensively studied snakebite-induced AKI and reported elevated urea and creatinine levels as common findings. Kumar et al. (2022) 19 also highlighted the significance of AKI as a major complication of snakebite envenomation.

Liver Function Parameters

The liver function parameters in this study revealed a mean total bilirubin level of 0.957 (SD = 0.667) and a mean direct bilirubin level of 0.407 (SD = 0.493). The mean AST and ALT levels were 46.11 (SD = 42.02) and 44.42 (SD = 31.14), respectively. The mean LDH level, measured in 37 patients, was 484.38 (SD = 602.132). These findings suggest that snakebite envenomation can also affect liver function, although the extent of liver involvement may vary. Elevated bilirubin and liver enzymes can indicate hepatocellular damage or cholestasis, which may contribute to the overall morbidity associated with snakebites.

ASV Administration

In this study, the majority of patients (92%) received Anti-Snake Venom (ASV), while a small proportion (8%) did not. ASV is the mainstay of treatment for systemic snakebite envenomation, and its timely administration is crucial for neutralizing venom toxins and preventing or mitigating complications. The high percentage of ASV administration in this study reflects the adherence to established treatment protocols. However, variations in ASV administration rates can occur due to factors such as the severity of envenomation, the time of presentation, and the availability of ASV. Studies like Sujir et al. (2018) 13 and B R et al. (2019) 20 have emphasized the importance of early ASV administration in improving patient outcomes. The decision not to administer ASV in some cases might be due to early recovery, mild envenomation, or contraindications. The impact of ASV administration on specific outcomes, such as the development of TMA and AKI, will be discussed in subsequent sections.

AKI Development

In this study, 39% of patients developed Acute Kidney Injury (AKI). This incidence falls within the wide range of AKI prevalence reported in the literature following snakebite envenomation. Studies have shown AKI incidence varying from 8% to as high as 60% as mentioned by Sarkar et al. (2021) 21. For instance, Rao et al. (2024) 22 reported an 18.5% incidence of TMA-associated AKI, while Kumar et al. (2022) 19 found an AKI incidence of 20.7% in snake envenomation. The variability in AKI incidence across studies can be attributed to differences in geographical location, snake species involved, the severity of envenomation, and the criteria used to define AKI. The relatively high incidence of AKI in our study underscores its importance as a major complication of snakebite, contributing significantly to morbidity and mortality.

Dialysis Requirement

Among the patients who developed AKI in this study, 59% required dialysis. This highlights the severity of renal involvement in a substantial proportion of snakebite-associated AKI cases. Other studies have also reported on the need for renal replacement therapy (RRT) in snakebite patients with AKI. Rao et al. (2019) 18 found that 95% of AKI patients with TMA required renal replacement therapy, indicating a higher dialysis requirement in TMA-associated AKI.

Schistocytes in Peripheral Smear

In this study, all patients with TMA (100%) had schistocytes present in their peripheral blood smear. The presence of schistocytes is a hallmark of microangiopathic hemolytic anemia (MAHA), a key component of TMA. The definition of snakebite-associated TMA often includes the presence of MAHA, along with thrombocytopenia. Noutsos et al. (2022) 23 proposed diagnostic criteria for snakebite-associated TMA as anemia with $>1.0\%$ schistocytes on blood film examination, together with absolute thrombocytopenia ($<150 \times 10^9/L$) or a relative decrease in platelet count of $>25\%$ from baseline.

Renal Biopsy Findings

Among the patients who underwent renal biopsy in this study, 88.9% showed fibrinoid necrosis or intraglomerular thrombi deposition, while 11.1% had no pathological findings. Fibrinoid necrosis and thrombi deposition are characteristic features of TMA, indicating microvascular injury and thrombosis within the kidneys. Vikrant et al. (2017) 17 also examined renal pathology in snakebite-associated AKI and reported acute tubular necrosis (ATN) as the predominant lesion. While ATN is a common finding in AKI from various causes, the presence of fibrinoid necrosis and thrombi strongly suggests TMA as the underlying mechanism of kidney injury. These pathological findings provide valuable insights into the pathophysiology of renal involvement in snakebite-associated TMA, highlighting the role of microvascular damage and coagulation abnormalities.

Development of TMA

In this study, the overall incidence of thrombotic microangiopathy (TMA) was 9%. This finding is within the range of TMA prevalence reported in other studies, although there is considerable variation. For instance, Mohan et al. (2019) 24 reported a TMA prevalence of 18.8% in their study conducted in Karnataka, India, which is higher than the incidence observed in our study. Conversely, Noutsos et al. (2022) 16 found an overall prevalence of TMA in envenomed patients to be 9%, consistent with our finding, but with variations depending on the snake species.

Association of Demographics and Snakebite Characteristics with TMA

The analysis of the association between demographic and snakebite characteristics with the presence of TMA in this study revealed no statistically significant associations with age group, gender, snake type, or time to presentation ($p > 0.05$ for all Chi-square tests). This finding

contrasts with some previous studies that have identified risk factors for TMA or AKI following snakebites. 18

Association of Clinical Features with TMA

The strong association of oliguria/hematuria with TMA is consistent with the understanding that AKI is a common and severe complication of TMA. Several studies have highlighted the close relationship between TMA and AKI 16, 18.

Comparison of Hematological Parameters with TMA

The comparison of hematological parameters between patients with and without TMA in this study revealed significant differences in PT, INR, Hb, and platelet counts. Patients with TMA had significantly higher PT and INR values, indicating coagulopathy, and significantly lower Hb and platelet counts, reflecting microangiopathic hemolytic anemia (MAHA) and thrombocytopenia, the hallmarks of TMA. These findings align with the pathophysiology of TMA, which involves microvascular injury, platelet activation, and hemolysis. Studies defining TMA^{23,16}, have consistently emphasized the presence of MAHA and thrombocytopenia as key diagnostic criteria

Conclusion:

This study provides evidence that thrombotic microangiopathy (TMA) is a notable complication following snake bite envenomation, observed in 9% of the studied patients. The occurrence of TMA was significantly associated with specific clinical manifestations, notably oliguria/haematuria and jaundice, as well as with distinct haematological abnormalities, including derangements in PT, INR, WBCT, and platelet counts

References

1. Afroz A, Siddiquea BN, Chowdhury HA, Jackson TN, Watt AD. Snakebite envenoming: A systematic review and meta-analysis of global morbidity and mortality. Habib AG, editor. PLoS Negl Trop Dis. 2024 Apr 4;18(4):e0012080.
2. GBD 2019 Snakebite Envenomation Collaborators, Roberts NLS, Johnson EK, Zeng SM, Hamilton EB, Abdoli A, et al. Global mortality of snakebite envenoming between 1990 and 2019. Nat Commun. 2022 Oct 25;13(1):6160.
3. Waiddyanatha S, Silva A, Siribaddana S, Isbister GK. Long-term Effects of Snake Envenoming. Toxins. 2019 Mar 31;11(4):193.
4. Chippaux JP. Snakebite envenomation turns again into a neglected tropical disease! J Venom Anim Toxins Incl Trop Dis. 2017 Dec;23(1):38.
5. Suraweera W, Warrell D, Whitaker R, Menon G, Rodrigues R, Fu SH, et al. Trends in snakebite deaths in India from 2000 to 2019 in a nationally representative mortality study. eLife. 2020 Jul 7;9:e54076.
6. Senji Laxme RR, Khochar S, De Souza HF, Ahuja B, Suranse V, Martin G, et al. Beyond the 'big four': Venom profiling of the medically important yet neglected Indian snakes reveals disturbing antivenom deficiencies. Billiald P, editor. PLoS Negl Trop Dis. 2019 Dec 5;13(12):e0007899.
7. Palangasinghe DR, Weerakkody RM, Dalpatadu CG, Gnanathanan CA. A fatal outcome due to pulmonary hemorrhage following Russell's viper bite. SMJ. 2015 May;36(5):634-7.
8. Afroz A, Siddiquea BN, Shetty AN, Jackson TNW, Watt AD. Assessing knowledge and awareness regarding snakebite and management of snakebite envenoming in healthcare workers and the general population: A systematic review and meta-analysis. Monteiro WM, editor. PLoS Negl Trop Dis. 2023 Feb 9;17(2):e0011048.
9. Noutsos T, Currie BJ, Lek RA, Isbister GK. Snakebite associated thrombotic microangiopathy: a systematic review of clinical features, outcomes, and evidence for interventions including plasmapheresis. Gutiérrez JM, editor. PLoS Negl Trop Dis. 2020 Dec 8;14(12):e0008936.
10. Ihsan H, Raina S, Raina RK, Sharma R. Prevalence of Thrombotic Microangiopathy Among Patients With Snake Bite-Induced Hemotoxic Clinical Syndrome in the Hills of Himachal Pradesh, India. Medical Journal of Dr DY Patil Vidyapeeth. 2024 Mar;17(2):322-7.
11. Sasidharan P, Kaeley N, Mahala P, Jose JR, Shankar T, Santhalingan S, et al. Clinical and demographic profiling of snakebite envenomation in a tertiary care centre in northern India. Int J Emerg Med. 2025 Mar 10;18(1):50.
12. Chandrakumar A, Suriyaprakash TNK, Mohan PL, Thomas L, Vikas PV. Evaluation of demographic and clinical profile of snakebite casualties presented at a tertiary care hospital in Kerala. Clinical Epidemiology and Global Health. 2016 Sep;4(3):140-5.
13. Sujir SN. A Study of Clinical Presentation, Coagulation Profile, and Outcome of Vasculotoxic Snake Bite Envenomation in a Tertiary Care Hospital [PhD Thesis]. Rajiv Gandhi University of Health Sciences (India); 2018.
14. Alvitigala BY, Gooneratne LV, Gnanathanan CA, Wijewickrama ES. Snakebite-associated acute kidney injury in South Asia: narrative review on epidemiology, pathogenesis and management. Transactions of The Royal Society of Tropical Medicine and Hygiene. 2025 Jan 3;trae077.
15. Isbister GK, Little M, Cull G, McCoubrie D, Lawton P, Szabo F, et al. Thrombotic microangiopathy from Australian brown snake (Pseudonaja) envenoming. Internal Medicine Journal. 2007 Aug;37(8):523-8.
16. Noutsos T, Currie BJ, Isoardi KZ, Brown SG, Isbister GK. Snakebite-associated thrombotic microangiopathy: an Australian prospective cohort study [ASP30]. Clinical Toxicology. 2022;60(2):205-13.
17. Vikrant S, Jaryal A, Parashar A. Clinicopathological spectrum of snake bite-induced acute kidney injury from India. WJN. 2017;6(3):150.
18. Rao I, Prabhu A, Nagaraju S, Rangaswamy D. Thrombotic microangiopathy: An under-recognised cause of snake-bite-related acute kidney injury. Indian J Nephrol. 2019;29(5):324.
19. Kumar M, Arcot Thanjan M, Gopalakrishnan N, Jeyachandran D, Thanigachalam D, Ramanathan S. Snake envenomation-induced acute kidney injury: prognosis and long-term renal outcomes. Postgraduate Medical Journal. 2022 Apr 1;98(1158):264-8.
20. BR MM. Study of Clinical Features of Snake Bite and its Prognosis [PhD Thesis]. Rajiv Gandhi University of Health Sciences (India); 2019.
21. Sarkar S, Sinha R, Chaudhury AR, Maduwage K, Abeyagunawardena A, Bose N, et al. Snake bite associated with acute kidney injury. Pediatr Nephrol. 2021 Dec;36(12):3829-40.
22. Rao PSK, Priyamvada PS, Bammigatti C. Snakebite envenomation-associated acute kidney injury: a South-Asian perspective. Transactions of The Royal Society of Tropical Medicine and Hygiene. 2025 Jan 3;trae114.
23. Noutsos T, Currie BJ, Wijewickrama ES, Isbister GK. Snakebite Associated Thrombotic Microangiopathy and Recommendations for Clinical Practice. Toxins. 2022 Jan 14;14(1):57.
24. Mohan G, Guduri PR, Shastry S, Kandasamy D. Thrombotic microangiopathy in hematoxic snakebites and its impact on the prognosis: an entity often overlooked. J Thromb Thrombolysis. 2019 Oct;48(3):475-82.