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Cardiovascular Disease Events in Hemotoxic Snakebite Envenoming: A Prospective Observational Study from Himachal Pradesh, India

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Abstract

Background: Cardiotoxicity as a feature of snake bite envenomation toxidrome is uncommonly described. The study was conducted to assess the prevalence of cardiovascular disease events in patients with hemotoxic venomous snakebites.

Material and Methods: This was a hospital based open cohort observational study conducted on patients diagnosed with hemotoxic envenoming. The study period was of one year using a nonprobability sampling method. Hemotoxic envenoming was defined as positive bedside 20 min whole blood clotting time (20 WBCT) following a history of snake bite. Cardiovascular events were recorded based on clinical history and examination, electrocardiography, high sensitivity (HS) troponin I levels and echocardiogram. Time frames to understand the sequential changes during the hospital stay were defined as less than 48 h, 48 to 96 hours, and at discharge.

Results: A total of 62 patients were included in the study. The prevalence of cardiovascular disease events was 16.1% (10/62). ECG changes were observed in nine patients. Three patients developed acute coronary syndrome, One patient had features of capillary leak syndrome. presented with shock and required inotropes.

Conclusion: The study demonstrated that cardiovascular disease events are not an uncommon entity due to hemotoxic snakebite envenoming in the geographical region of Himachal Pradesh, India.

Keywords: Cardiac, heart, myocardial infarction, snakes

Introduction

The common toxidromes due to snakebite envenoming (SBE)includes local envenoming with hemotoxicity, local envenoming with neuroparalysis, neuroparalysis with minimal or no local envenoming, local envenoming with hemotoxicity and acute kidney injury. Other short-term life-threatening systemic complications include cardiovascular, capillary leak syndrome, thrombotic microangiopathy and pituitary insufficiency.[1] Cardiovascular complications are uncommon. Transient ECG changes and acute myocardial infarction are the commonest cardiovascular complication particularly after Viperidae envenoming. In addition, hypotension, arrhythmias, pulmonary edema, myocarditis and stress cardiomyopathy have been reported in the literature.[2] The management of acute coronary syndrome in patients with hemotoxic envenoming is clinically challenging. The phenomenon of coagulopathy and thrombocytopenia limits the pharmacological and invasive approaches of coronary artery disease. In the setting of SBE, acute myocardial infarctions are mostly type 2, and in absence of coronary angiograms the post infarction treatment approaches are debatable. Performing contrast studies like coronary angiography and CT angiography in patients with SBE and acute kidney injury is another challenge. Epidemiology measurements on the burden of cardiovascular complications of SBE are limited. The Indian federal state of Himachal Pradesh has a distinct Viperidae snake fauna and includes Northern white lipped pit viper (Trimeresurus septentrionalis), Himalayan pit viper (Gloydiushimalayanus), Chamba pit viper (Gloydiuschambensis) in addition to Russell's viper (Daboia russelli) and saw scaled viper (Echis carinatus). The local distinct reptilian fauna makes it imperative to study the spectrum of envenoming from this geographic region. The objective of this study was to determine the prevalence of cardiovascular disease events among patients with hemotoxic envenoming admitted in a medical college hospital of Himachal Pradesh, India.

Materials and Methods

This was a hospital-based open cohort prospective descriptive study. The recruitment period was one year between June 2023 to May 2024 using a nonprobability sampling method. The inclusion criteria were patients above the age of 18 years presenting with hemotoxic envenomation following a history of snakebite. Those patients who were known to have pre-existing kidney disease, chronic liver disease, hypothyroidism and heart failure were excluded from the study. Patients discharged within 48 hours of admission were also excluded from the study.

Definitions

Hemotoxic envenomation

It was defined as positive 20 WBCT following a history of snake bites.[3]

Cardiovascular disease events

Cardiovascular events were recorded based on clinical history and examination, electrocardiography, HS-troponin I leveland echocardiogram. They were defined as any of the following. A history suggestive of new onset cardiac disease, ECG abnormalities, elevation of HS- troponin I level, echocardiographic changes, hypotension (systolic blood pressure less than 90 mmmHg) and inotropic support.[4,5]

Procedure

Patients recruited to the study had demographic, clinical details, and treatments recorded. All the recruited patients had complete hemograms, peripheral smears, liver function tests, renal function tests, and urine examinations. ECG was done in all patients. Patients with ECG changes suggesting acute coronary syndrome were subjected to HS-Troponin I estimation. Patients with raised troponins were evaluated by echocardiogram. Time frames to understand the sequential changes during the hospital stay were defined as less than 48 h, 48 to 96 hours, and at discharge. The peak value at the time of clinical manifestation was taken for the diagnosis. Data were entered in the Microsoft office excel sheet and analyzed through Epi-info-7.Quantitative variables were expressed as mean with standard deviation and categorical variables as frequencies and percentages. Median ± IQR was calculated for data with uneven and wide distribution and extreme values. The study was approved by the institutional ethics committee of Dr. Rajendra Prasad Govt Medical College, Tanda vide no HFW-H DRPGMC/Ethics/2023/016 dated 6-04-2023. On the days of data collection, the nature and the purpose of the study were briefed to the participants. They were assured about the confidentiality and anonymity of the information collected. Informed written consent was taken.

Results

In this study, 62 patients were recruited over a period of one year. All the patients belonged to the areas located in Shivalik and the Lesser Himalayan region of Himachal Pradesh, India. All the subjects were diagnosed with hemotoxic envenoming. All the patients received Indian polyvalent snake antivenom to a maximum of 300 ml. None of the patients experienced an adverse reaction to the antivenom. The demography, duration of admission,

and complications are shown in Table 1. Cardiotoxic events were observed in 16.1% (10/62) patients as shown in Table 2. The patients included 9(14.5%) were having ECG changes. A 25-year male patient presented with hypotension following Russell viper bite and developed features of capillary leak syndrome. He was managed with inotropes. The major cardiac events were ECG changes and their distribution is shown in Table 3. A comparison of demography, clinical features, investigations and outcome in patients with and without cardiovascular events is shown in Table 4. It showed statistical indifference for all the parameters, and statistical difference was observed for lactate dehydrogenase only. Among the nine patients with ECG changes, six patients had either ST depression or T wave inversion and normal HS troponin levels. Rest three patients presented with features of acute coronary syndrome. Among these three patients two developed typical chest pain. All three patients had significantly raised HS troponins and regional wall motion abnormalities on echocardiogram. None of the three patients reported any significant past history. All the three patients had positive 20WBCT at admission. The coagulation profile was deranged in all three cases as shown in Table 5. All the three cases received medical management by single antiplatelet(aspirin), ACE inhibitor (ramipril), beta blocker(bisoprolol), high dose statin (rosuvastatin) and oral nitrates.None of the patient received anticoagulants. Patient 1 was a smoker. He had a snake bite on 14-8-2023 at 2 pm. Hewas admitted in a local hospital where he received 150 ml ASV. His baseline ECG on 15-8-2023 is shown in Figure 1 A. He developed typical chest pain associated with profuse sweating on 17-8-2023. His ECG at 5 am on 17-8-2023 at local hospital is shown as Figure 1 B. He was referred to our hospital. The ECG at our hospital at 9 am on 17-8-2023 is shown as Figure1C.He had an ongoing chest pain. Decision against primary PCI was taken after consultation with the cardiology department of our hospital. His pain improved with medical management. He was discharged on dual antiplatelets. Patient was advised coronary angiography at the time of discharge and during follow-up by the cardiology department but due to financial difficulties could not afford it. Patient 2 developed. typical chest pain, 14 hours after snakebite. He had evidence of anterior wall NSTEMI (Figure 1 D). He was medically managed which included single antiplatelet and dual antiplatelets were prescribed at discharge. Patient 3, a female presented with features of atypical chest pain at admission. She had evidence of anterolateral wall NSTEMI (Figure 1 E). Her platelet count was 23,000 on admission. She was medically managed which included single antiplatelet which was started on day three of admission when her platelet count was 81,000. Her platelet count at discharge was 117000 and she was prescribed dual antiplatelets. The acute myocardial infarction couldn't be classified into any particular type according to the clinical circumstance as none of the patient underwent coronary angiography and follow-up echocardiogram. Six other patients with either ST depression or T wave inversion had normal HS troponin levels. No death was observed among the cohort recruited in this study.

Figure 1

(A) The ECG of patient 1 at local civil hospital on 15-8-2023.
(B)The ECG of patient 1 on 17-8-2023(5am) at local civil hospital.
(C) The ECG of case 1 on 17-8-2023(9am) at our hospital.
(D) The ECG of patient 2 showing T wave inversion in V3-V6.
(E) The ECG of patient 3 showing T wave inversion I, AVL, V2-V6.

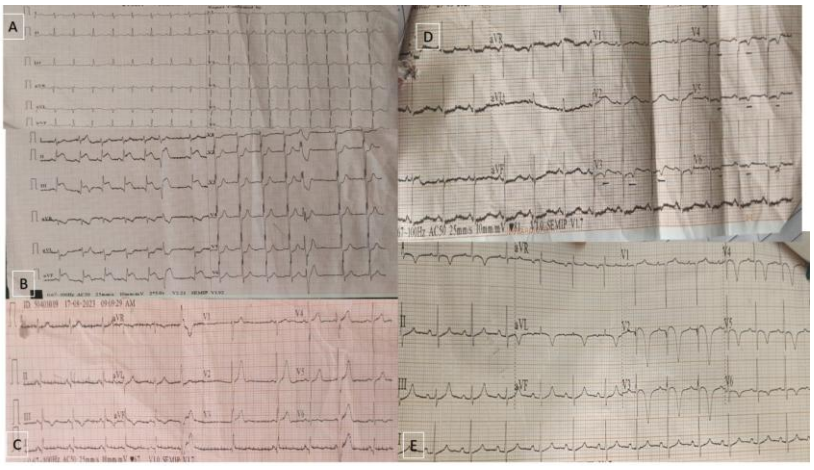

Table 1. Showing demography, duration of admission and complications among study subjects

Parameter Frequency distribution	(n=62)
Sex	
Males	35(56.5%)
Females	27(43.5%)
Mean age (years±SD)	43.2±16.2
Median time duration of snakebite to admission in hours (IQR)	4(2-12)
Median duration of admission of patients in days(IQR)	4(3-5)
Median duration for which 20 min WBCT remained positive in hours(IQR)	16(12-24)
Acute kidney injury	25(40.3%)
Capillary leak syndrome	12(19.3%)
Thrombotic microangiopathy	4(6.4%)

Table 2. Showing distribution of cardiotoxic events

Cardiotoxicity	Frequency distribution (n=62)
ECG changes	9(14.5%)
ECG changes with normal HS Troponin	6(9.6%)
ECG changes with raised HS Troponin I and RWMA on echocardiogram	3(4.8%)
Hypotension with inotropic support1(1.6%)	
Total	10(16.1%)

Table 3. Showing distribution of ECG changes

ECG Parameter	Frequency distribution (n=62)
Normal	53(85.5%)
Sinus bradycardia	2(3.2%)
ST depression II,III	1(1.6%)
ST elevation II,III,AVF;ST depression in I,AVL,V1-V6	1(1.6%)
T wave inversion II,III,AVF,V3-V6	1(1.6%)
T wave inversion I, AVL ,V2-V6	1(1.6%)
T wave inversion V1-V4	1(1.6%)
T wave inversion V1-V6,III,AVF	1(1.6%)
T wave inversion V3-V6	1(1.6%)

Table 4: Comparison of demography, clinical features, investigations and outcome in patients with and without cardiovascular events

Parameter	CVD events absent (n=52)	CVD events present (n=10)	pvalu p-value
Sex distribution			
Male	28	7	0.49
Female	24	3	
Age(years)	41.6 + 15.9	51+ 11.4	0.06
Median (IQR) time duration of snakebite to admission in hours	4(2-6)	3.5(2-15)	0.75
Tourniquet applied			
Yes	38	7	0.84
No	14	3	
Median (IQR) time duration of tourniquet application in minutes	37.5(18.7-66.2)	30(15-110)	0.95
Median (IQR) duration for which 20 min WBCT remained positive in hours	13(11.5-24)	15 (10.5-24)	0.84
Total ASV dose received			
100 mL	11	0	0.07
200 ml	17	3	0.54
250 mL	1	2	0.26
300 mL	23	5	0.12
20WBCT normalized in(hours)	224±78	260±45	0.16
Median (IQR) duration of admission in days	4(3-5)	5(3-5)	0.30
Haemoglobin (gm/dl)	10.5±1.9	10.7±2.1	0.82
TLC(per µL)	14128±5365	15490±3328	0.44
Platelet (per µL)	88211±39153	84400±39900	0.78
Urea(IQR),mg/dl	40(27.5-81.2)	45(33.7-98.5)	0.42
Creatinine (IQR),mg/dl	1.1(0.7-1.9)	4.9(1-3)	0.22
AST(IQR),U/L	52(39-95)	86(52-247)	0.12
ALT(IQR),U/L	31(21-74)	47(23-106)	0.23
ALP(U/L)	105±41	100±28	0.71
S. Albumin(gm/dl)	3.4±0.4	3.4±0.5	0.98
LDH(IQR),U/L	452(342-916)	1208(648-1995)	0.01
Total CPK(µg/L)	320(146.2-567)	539(137-735)	0.55
Uric acid(mg/dl)	6±1.5	6.2±1.6	0.84
Capillary leak syndrome 9	3	0.39	
Acute kidney injury 20	5	0.50	
Dialysis	9	2	1.00
Outcome			
Improved	50	10	1.00
Expired	2	0	

n-number; IQR- interquartile range; ASV- antisnake venom; 20WBCT- 20 minute whole blood clotting test; TLC- total leucocyte count; AST-aspartate transaminase; ALT-alanine transaminase; ALP-alkaline phosphatase; LDH-lactate dehydrogenase; CPK-creatine phosphokinase

Table 5. Showing the profile of three cases with acute coronary syndrome

Profile	Patient 1	Patient 2	Patient 3
Age (years)	51	66	62
Sex	Male	Male	Female
Time of snake bite	2PM	12:10 AM	12:00AM
Bite to onset of chest pain	48 hours	14 hours	30 minutes
Symptoms	Typical chest pain Diaphoresis	Typical chest pain epigastric discomfort Nausea, diaphoresis	Atypical chestpain
Snake identified as	Northern white lipped Pit viper	Russells viper	Russells viper
ECG changes inversion	ST segment elevation II,III,AVF	T wave inversion V3-V6	T wave I,AVL, V2-V6
HS TROP I	22.1ng/ml (<0.03)	172pg/ml(<20)	458pg/ml(<20)
Echocardiogram	RWMA Inferior wall	RWMA lateral wall	RWMA apicolateral wall
ASV given	250ml	300ml	300 ml
20-WBCT normalised in	96 hours	26 hours	18 hours
Length of hospital stay	10 days	3 days	4 days
Platelet count (perµl)	161000	99,000	23,000
PT/INR	18.8/1.6	16.1/1.5	17.9/1.5
APTT 45.6	25.2	24.2	
D-Dimer (µg/ml)	10	10	10
AST (U/L)	259	93	30
ALT (U/L)	70	23	27
Creatinine (mg/dl)	0.8	1.2	0.7
CPK(µg/L)	666	not done	not done
LDH (U/L)	1283	not done	not done

RWMA- regional wall motion abnormalities; ASV- antisnake venom; WBCT- whole blood clotting test; PT- prothrombin time; INR- international normalized ratio; APTT- activated partial thromboplastin time

Table 6. Summary of studies describing cardiac complications in snakebite envenomation and comparison with the present study

Study	Type of study(number)	location	cardiac manifestation
Cupo et al[16]	Prospective (7)	Brazil	First-degree AV block (n = 1); junctional pacemaker (n=1); tall T waves (n=2); U waves (n=1)
Karlson-Stiber et al[17]	Retrospective (231)	Sweden	ECG changes (including ST depression, T-wave inversion, T wave flattening, atrial fibrillation, ventricular extrasystoles, and atrio-ventricular block grade I) (n =8); myocardial infarction (n =1)
Suchitra et al[18]	Prospective (586)	India	Ventricular tachycardia(n=1)
Kim et al[19]	Retrospective (98)	South Korea	Myocardial infarction (n = 1)
Magdalan et al[20]	Case series (26)	Poland	Hypotension (n =4); Supraventricular arrhythmia (n =2)
Honge et al[21]	Prospective (586)	Denmark	T wave inversion(n=5)
Kim et al[12]	Retrospective (65)	South Korea	T-wave inversion (n = 2); QT prolongation (n = 4)
Johnston et al[22]	Prospective (40)	Australia	Ventricular fibrillation cardiac arrest followed by bradycardic arrest (n = 1)
Ramakrishna et al[14]	Prospective (200)	India	Sinus tachycardia(n=27); Sinus bradycardia(n=6) Ventricular tachycardia (n=1)
Binu et al[5]	Retrospective (34)	India	Prolonged QTc (62.5%), T-wave inversion (37.5%) and tall T-waves (12.5%)
Sunil et al[4]	Prospective (96)	India	ECG changes in 34.3% and rise in troponin-I in 21.9% of patients, echocardiographic changes in 4.2%, and Takotsubo cardiomyopathy in 1%.
Current study	Prospective (62)	India	ECG changes with normal HS Troponin(n=6); ECG changes with raised HS Troponin I and RWMA on echocardiogram(n= 3); hypotension with inotropic support(n=1)

Discussion

Cardiotoxicities are not common complications of snakebite envenomation (SBE) but they do occur. Information on cardiotoxicity among patients with snakebite envenoming is largely limited to tropical countries of India and Sri Lanka from southeast Asia, South Korea in East Asia and Nigeria in West Africa.[4,13] In India, data on cardiac complication in SBE is available from the states of Kerala, Tamil Nadu, Rajasthan, Jammu and West Bengal.[4-8,14,15]

Patients recruited in the present study had hemotoxic envenoming diagnosed on the basis of positive 20WBCT. The prevalence of cardiotoxicity was 16.1% among patients in this study. In a study south Indian state of Kerala comprising 96 study subjects of vasculotoxic and neurotoxic snakebite the cardiac toxicity was observed in 42.7% of patients. The ECG changes, rise in troponins, echocardiographic changes and Takotsubo cardiomyopathy were observed in 34.3%, 21.9%, 4.2%, and 1% respectively. In the same study, difference between the neurotoxic (41.7%) and vasculotoxic (42.9%) snake bites induced cardiotoxicity was not statistically different.[4]In another retrospective study from south Indian state of Tamil Nadu cardiovascular effects were observed in 70.6% of cases. The commonest ECG changes observed were prolonged QTc (62.5%), T-wave inversion (37.5%), tall T-waves (12.5%) and ST elevation (4.1%).[5]In a retrospective Korean study, the prevalence of adverse cardiovascular events following SBE was 13.8%.[12] In a study of 108 patients with Viperidae snakebite fromNigeria, electrocardiographic abnormalities and raised troponins were observed in 60% and 2% patients respectively.[13] In a systematic review of case reports, prospective and retrospective studies the prevalence of cardiovascular compromise was 0.2%-3.8%.[2] The subject on the prevalence of cardiovascular complications of SBE have shown a wide variation in the measurement frequency across geographical regions. A review of the observations made in the studies published since 2003 is shown in Table 6. The vast variation is possibly because of the unavailability of robust information as the analysis is from both retrospective and prospective studies. In addition, the cardiovascular manifestations are heterogenous and the definition of cardiotoxicities has not been standard in these observations.

The description of acute coronary syndrome is largely expressed in case reports.[9,10,23,26] The description of acute myocardial infarction is of both type 1 and type 2. For the definition of type 1 acute myocardial infarction, coronary angiograms have documented thrombus in coronary arteries which have been managed by primary angioplasty and placement of bare metal stent.[27,29] Type 2 acute myocardial infarction as by evidence of imbalance between myocardial oxygen supply and demand unrelated to acute atherothrombosis has been documented by normal coronary arteries on coronary angiography in some of these patients after thrombolysis with reteplase andtenecteplase.[15,30] Fibrinolytic agents reteplase and tenecteplase has been used in ST segment elevated myocardial infarction (STEMI) without precipitating coagulopathy.[15,30] GP2b/3a inhibitor tirofiban infusion was used in one of a patient without affecting coagulopathy.[28] Dual antiplatelets, aspirin and clopidogrel has been prescribed in patients with normal platelet count and acute coronary syndrome. [30,31]There is no evidence on the safety of antiplatelet therapy in patients with thrombocytopenia and acute coronary syndrome in the setting of snake envenomation. Optimal antiplatelets strategy in patients with thrombocytopenia and acute coronary syndrome is a matter of research and debate. Researchers are of the opinion the clopidogrel as a single antiplatelet should be prescribed in patients with platelet count between 50×10^9/L to 100×10^9/L.[32] CT coronary angiography has been performed in a patient with acute myocardial infarction with and without heart failure.[11,31] This imaging modality is rational as most of the patients are young and have a low risk for ASCVD. This imaging modality will provepractically useful in deciding about continuation of antiplatelet drugs in these patients. Six patients had ECG changeswith normal HS troponins in our study. The possible reason for ST depression and T wave inversion can be due to hyperventilation, ventricular strain or adrenergic stress.

Evidence on the diagnostic reliability of cardiac troponins in the setting of snakebite is limited to patients with symptoms of acute coronary syndrome. Cardiac troponin (cTn) I or T (cTnI or cTnT) are the biomarker of choice for the detection of myocardial injury. Myocardial injury is caused by a variety of noncardiac and cardiac conditions other than acute myocardial ischemia. The diagnostic reliability of cardiac troponins has been suboptimal in patients with acute kidney injury, chronic kidney disease and after strenuous exercise.[33] Muscle injury and acute kidney injury is a known complication of snakebite envenomation. A PubMed search with keywords ‘snake envenoming’; ‘troponins’ did not yield any results other than in patients with cardiac involvement. Animal model assays have been performed for serum creatine kinase skeletal muscle troponin-I concentration, skeletal muscle troponin-C concentration, myoglobin activity, skeletal muscle myosin light chain-1 concentration, and creatine kinase-MM activity for detection of myotoxic snake envenoming.[34]

The outcome of patients with acute myocardial infarction and SBE is variable and fatalities are also reported.[9,10]

Viper venoms are composed of three dominant protein families of snake venom metalloprotease (SVMP), phospholipase A2 (PLA2) and snake venom serine protease (SVSP). The secondary protein families include disintegrins (DIS), cysteine-rich secretory protein (CRiSP), C-type lectins (CTL), L-amino acid oxidase (LAAO), kunitz peptides (KUN), and natriuretic peptides (NP).[35]

The exact mechanisms of cardiac manifestations are unknown. Thrombotic microangiopathic hemolysis, direct effects of venom myotoxins on myocardium, venom-induced coronary artery spasm, thrombosis, hypovolemia due to anaphylactic shock or bleeding, electrolyte disturbances, stress induced hyper catecholaminaemia (Takotsubo cardio myopathy) and Kounissyndrome are the possible causes for cardiac involvement in these case reports.[11,36]

Presence of thrombocytopenia and coagulopathy poses a clinical challenge on the management of acute coronary syndrome in patients with SBE. No consensus guidelines are available to guide clinicians on the management of such patients. The evidences are limited hence the treatment should be individualized.

Strengths and Limitations

The study from the physio-geographic region of Himachal Pradesh India, is one of the few that provides a comprehensive characterization of the patient population of hemotoxic snakebite envenomation with cardiac events and comparing them with those who had no cardiac event. The limitations are that the patients with ST depression, T wave inversion and normal HS-troponins were not evaluated by echocardiograms. In addition,we were not able to perform either coronary angiograms or noninvasive CT coronary angiographyin patients with acute coronary syndrome. Follow-up electrocardiogram of patients with ECG changes and normal HS troponins were not recorded. Follow-up echocardiography of all the patients with ACS couldn't be performed.

Conclusion

Clinicians should be aware of adverse cardiovascular disease events in SBE. Threshold for screening and suspecting these events should be kept low and investigations like ECG, echocardiogram and troponins must be ordered with typical and atypical symptoms. Treatment of acute coronary syndrome can't be generalized and has to be individualized on case-to-case basis.

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Data availability statement

The data underlying this article are available in the article and will be shared upon reasonable request to the corresponding author.

Author contribution statement

AG and SR were involved in the study conception and study design. AG, SR, BS, MT, NK implemented the study and coordinated and conducted the experiments. All the authors were involved in interpreting the data. AG and SR wrote the first draft. All the authors read and approved the final version of the manuscript.

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Conflicts of Interest

None declared.

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