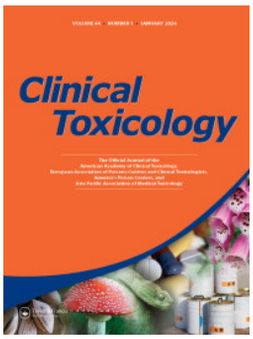

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POISON CENTRE RESEARCH

Characterization of anemia following rattlesnake envenomation in Arizona: a retrospective observational study

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ABSTRACT

Introduction: Rattlesnakes in the southwestern United States possess hemotoxic venom associated with coagulopathy and bleeding. While the two complications are related, they are distinct entities, and no known study has evaluated the phenomenon of venom-induced anemia.

Methods: A chart review was performed using electronic health records from the Arizona Poison and Drug Information Center between January 1st, 2017 and December 31st, 2022. Included charts were characterized by the presence or absence of anemia (hemoglobin <100 g/L), and clinical and demographic information were compared between the two subgroups.

Results: A total of 800 records were found within the time period, of which 705 patient records were included. Female sex was significantly associated with increased anemia rates (OR 2.23 [95% CI: 1.14–4.36]; $P=0.02$), but age was not (OR 0.99 [95% CI: 0.98–1.00]; $P=0.13$). When adjusting for age/sex, the prevalence of anemia was associated with dual anticoagulant/antiplatelet therapy (OR 55.4 [95% CI: 3.10-993]; $P=0.006$), visible inpatient bleeding (OR 10.1 [95% CI: 2.87–35.7]; $P<0.001$) and ecchymosis extending more than half an extremity (OR 6.17 [95% CI: 3.06–12.4]; $P<0.001$), among others.

Discussion: Anemia in the setting of rattlesnake envenomation is likely multifactorial, and potentially due to erythrocyte sequestration, occult bleeding, and erythrocyte destruction, both direct and indirect. Patients with anemia were shown to have higher rates of coagulopathy, specifically thrombocytopenia, and derangements to the international normalized ratio.

Conclusions: Patients on dual anticoagulant/antiplatelet or chronic non-steroidal anti-inflammatory drug therapy were found to have increased rates of anemia. Thrombocytopenia and an increased international normalized ratio were associated with higher rates of anemia when adjusting for age and sex.

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Introduction

Rattlesnakes of the southwestern United States (US) possess hemotoxic venom [1] that acts directly on coagulation factors necessary for maintaining hemostasis [2]. Although venom toxins cause both pro- and anticoagulating effects, envenomated patients commonly develop hypocoagulation [3]. In the US, the two available rattlesnake antivenoms, CroFab® and Anavip®, are both effective at neutralizing the toxins responsible for causing coagulopathy [4]. However, stopping the effects of venom toxins does not replace any of the lost clotting factors. It can take hours to days for a patient to synthesize a sufficient supply of new clotting factors [5], and some patients will remain profoundly hypocoagulable for prolonged periods of time. Like other hypocoagulable patients, envenomated patients are believed to be at an increased risk of bleeding complications should they sustain some form of mechanical traumatic injury. Although rare, the insidious development of unprovoked significant bleeding has also been reported [6].

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While hypocoagulability increases the likelihood of a patient bleeding, the two are distinct entities. The current literature describing venom-induced hemotoxicity fails to sufficiently distinguish between coagulopathy and bleeding. A definition of major bleeding for snakebite was suggested in the Global Core Outcome Measurement Set [7], namely fatal bleeding, symptomatic bleeding in a critical area/organ, or bleeding causing a fall in hemoglobin concentration by ≥20 g/L or requiring transfusion of ≥2 units of whole blood. However, a recent study found no fatal bleeding or symptomatic bleeding in critical areas/organs, and a ≥20 g/L drop in hemoglobin concentration was found to be nonspecific for rattlesnake envenomations in Arizona [8]. We are unaware of any prior studies focusing on venom-induced anemia. Given the potential consequences of critical bleeding and the overall lack of published guidance on the topic, an improved understanding of venom-induced anemia is needed to better inform treatment decisions.

Our objective is to compare pertinent demographic, laboratory, treatment, and outcome data of patients who developed anemia versus those who did not, following envenomation by a rattlesnake in Arizona.

Methods

A retrospective chart review was performed using Electronic Health Records of the Arizona Poison and Drug Information Center, which serves as the poison center for the state of Arizona, excluding Maricopa County. A search was performed for cases coded as rattlesnake bites where antivenom was administered between January 1, 2017, and December 31, 2022. Records were excluded when the patient was incarcerated, when poison center involvement in the patient care was incomplete, such as if patient care was transferred to another poison center, or if the poison center was not contacted until after the original hospital visit. A standardized study template was made for the remaining patient charts, containing basic demographic information and clinical data points.

Patient charts included in our study were categorized into two groups based on whether the patient developed anemia, defined as a hemoglobin concentration <100 g/L at any time [9]. Each group's demographic information and clinical data points were then compared. Laboratory derangements immediately after rattlesnake envenomation can often be outside the detectable range; for data analysis purposes, these values were changed to the reported cutoff value (e.g., international normalized ratio [INR] reported as >12 was changed to 12 for statistical analysis). To determine statistical significance, a χ^2 test was performed for discrete variables (e.g., the presence/absence of thrombocytopenia), and an unpaired t-test was performed for continuous variables (e.g., platelet nadir).

Multivariable logistic regressions were also performed to identify associations with anemia after rattlesnake envenomation. For regressions, continuous variables included age, total antivenom vials administered, platelet count nadir, fibrinogen concentration nadir, and INR peak. The following variables were considered categorical (factor) variables: sex, bite site (upper versus lower extremity), if antivenom was administered within 4 h of envenomation, anticoagulation/antiplatelet medications, non-steroidal anti-inflammatory drug use, type of antivenom received, if a fasciotomy was performed, antibiotic use, blister formation, if a debridement was performed, maximum extent of ecchymosis and edema progression from bite site while inpatient as reported by hospital staff, presence of visible bleeding, peak edema, pain level, functional impairment and the presence of increased INR values, thrombocytopenia and hypofibrinogenemia. Patients with both inpatient and outpatient bleeding were removed from the analysis due to the absence of anemia cases.

Statistical analysis was performed using R. Univariate logistic regressions were performed for age and sex with anemia as the outcome variable. Multivariable logistic regressions were also conducted individually for each predictor using the generalized linear model function after adjustment for age and sex. Because dosing schedules differ, modelling of the total number of antivenom vials was performed for patients treated with CroFab® versus Anavip® separately after adjustment for age and sex and exclusion of patients who received both antivenom products. An alpha level of 0.05 was used to establish statistical significance.

The Investigational Review Board at the University of Arizona approved this study.

Results

A total of 800 records were found within the time period of rattlesnake envenomated patients who received antivenom; 95 were excluded from the study (Figure 1). In total, 705 patient records were included in our study. Hemoglobin concentrations were obtained; 37 patients had a recorded hemoglobin concentration <100 g/L, meeting our criteria for anemia.

Table 1 contains the full demographic and outcome measures of both subgroups. Five of the 37 patients with anemia received blood products during their

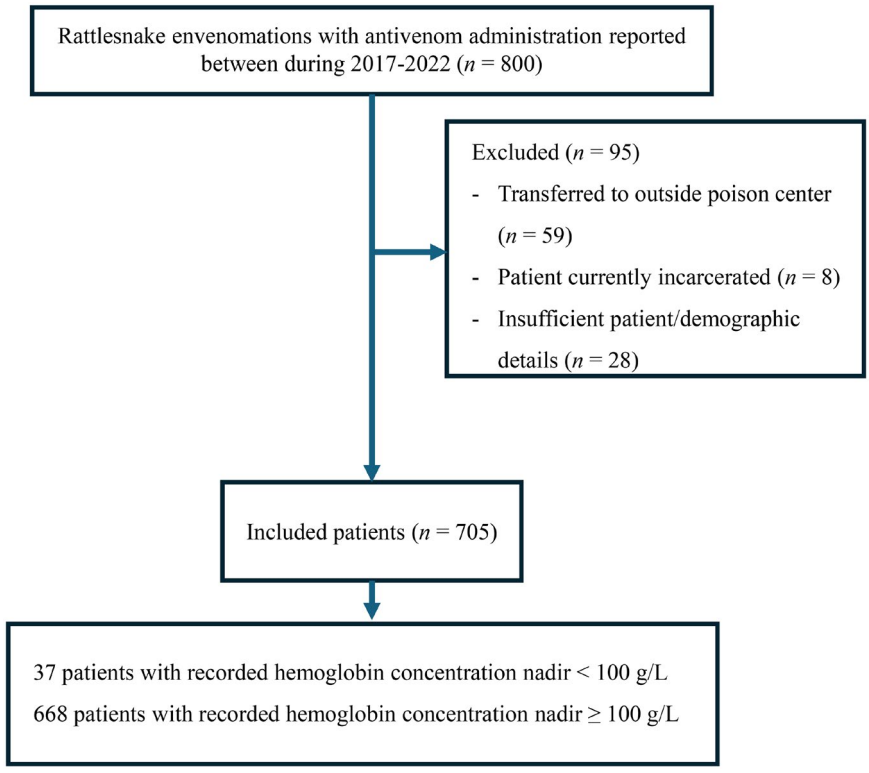

Figure 1. Inclusion/exclusion criteria.

Table 1. Characterization of venom-induced anemia.

Description	Total	Anemia	Without anemia	*P* value
Age, years				
Median (IQR)	53 (31–66)	58 (4–71)	53 (32–66)	0.124
Range	1–93	1–93	2–90	
Sex				
Male, *n* (%)	441 (63)	16 (43)	425 (64)	0.012
Home medications				
Anticoagulants, *n* (%)	32 (5)	4 (11)	28 (4)	0.060
Antiplatelet therapy, *n* (%)	11 (2)	2 (5)	9 (1)	0.053
Non-steroidal anti-inflammatory agents, *n* (%)	162 (23)	13 (35)	149 (22)	0.071
Anatomic location				
Upper extremity, *n* (%)	298 (42)	12 (32)	286 (43)	0.213
Lower extremity, *n* (%)	407 (58)	25 (68)	382 (57)	
Ecchymosis				
Peak > half extremity, *n* (%) 154 (22)		23 (62)	131 (20)	<0.001
Peak < half extremity, *n* (%) 551 (78)		14 (38)	537 (80)	
Laboratory observations				
Platelet count <150 x 10^9/L, *n* (%) 313 (44)		24 (65)	289 (43)	0.010
Fibrinogen concentration <1.5 g/L, *n* (%) 241 (34)		18 (49)	223 (33)	0.057
International normalized ratio ≥2.0, *n* (%) 91 (13)		10 (27)	81 (12)	0.009
Time to antivenom				
Start <4 h, *n* (%)	582 (83)	27 (73)	555 (83)	0.115
Start >4 h, *n* (%)	123 (17)	10 (27)	113 (17)	
Antivenom type[a]				
Crofab®, *n* (%)	412 (58)	18 (49)	394 (59)	
Anavip®, *n* (%)	344 (49)	20 (54)	304 (46)	
Transfusions[b]				
Packed red blood cells, *n* (%)	4 (1)	4 (11)	0	
Cryoprecipitate, *n* (%)	1 (0.1)	0	1 (0.1)	
Platelets, *n* (%)	1 (0.1)	0	1 (0.1)	
Multiple, *n* (%)	2 (0.3)	1 (3)	1 (0.1)	
Day 14 recovery				
Full, *n* (%)	179 (25%)	4 (11)	175 (26)	0.039
Partial, *n* (%)	211 (31%)	11 (30)	200 (30)	
Poor, *n* (%)	179 (25%)	16 (43)	163 (24)	
Missing, *n* (%)	136 (19%)	6 (16)	130 (20)	

[a]Antivenom type: numbers do not add to 100% because some patients received both products.
[b]Tranfusions: *P* value not calculated, given paucity of recorded transfusions.

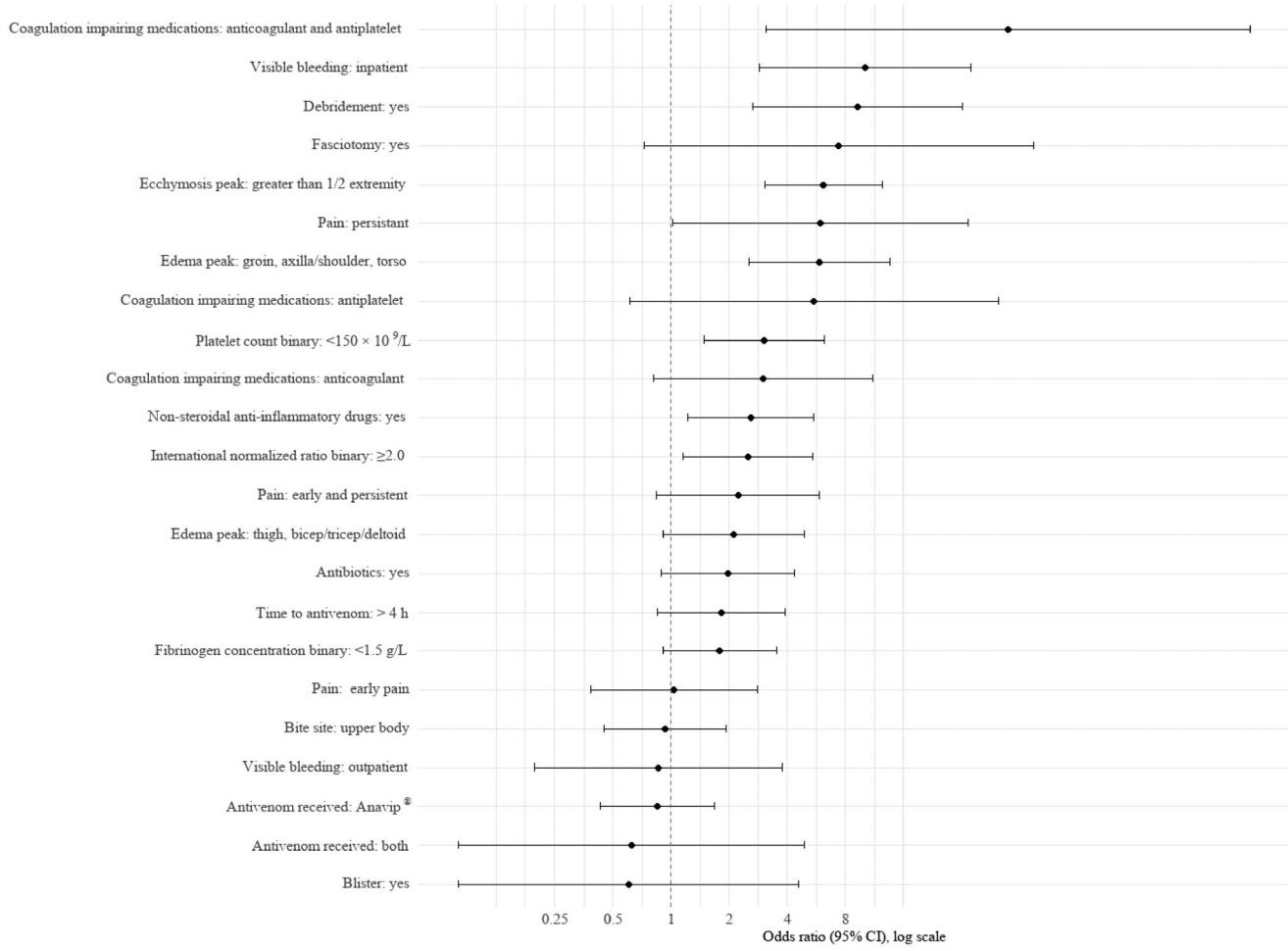

Figure 2. Forest plot of individual logistic models (adjusted for age and sex).

treatment course, compared to three of the 668 patients who did not develop anemia. Anemic patients were more likely to have ecchymosis spanning more than half an extremity. Likewise, anemic patients were more likely to have concurrent thrombocytopenia and an increased INR. Recovery rates, defined as the presence or resolution of pain, edema and functional impairment [10], 14 days after envenomation were worse in anemic patients.

Regression analyses revealed that female sex was significantly associated with a more than two-fold increase in anemia (OR: 2.23 [95% CI: 1.14–4.36]; $P = 0.02$), but age was not (OR: 0.99 [95% CI: 0.98–1.00]; $P = 0.13$) (Table 2). Logistic regressions adjusted for age and sex revealed multiple factors associated with anemia, including bites to the calf or thigh, non-steroidal anti-inflammatory drug use, patients on dual anticoagulant/antiplatelet therapy, if debridement was performed, ecchymosis extending past the nearest joint and into the torso, visible inpatient bleeding (defined as anytime between presentation to the emergency department and discharge from hospital), ongoing, severe pain despite repeated (three or more) doses of opioids [11], peak INR, an INR ≥2.0, and the presence of thrombocytopenia and platelet nadir (Table 3; Figure 2). Platelet nadir was significantly associated with a decrease in anemia risk. In patients treated with Crofab®, the total number of vials was significantly associated with anemia (OR: 1.19 [95% CI: 1.09–1.31]; $P = 0.0003$) (Table 4). In patients treated with Anavip®, the total number of vials was also significantly associated with anemia (OR: 1.11 [95% CI: 1.06–1.17], $P = 0.00002$). Hypofibrinogenemia was significantly associated with anemia in the unadjusted logistical regression (1.96 [1.01,3.81], $P = 0.047$); however, the association became insignificant after adjusting for age and sex (OR: 1.79 [95% CI: 0.909,3.51]; $P = 0.092$).

Discussion

Anemia following rattlesnake envenomation is potentially due to a variety of etiologies. The primary concern with coagulopathic patients is spontaneous or easily-provoked bleeding, which could result in anemia

Table 2. Univariate logistical regression for prediction of anemia.

Description	Odds ratio	95% confidence interval	*P* value
Demographics			
Age	0.99	(0.975–1.00)	0.134
Sex	2.23	(1.14–4.36)	0.019
Bite Site			
Upper extremity	0.71	(0.356–1.42)	0.334
Home medications			
Anticoagulants	2.19	(0.630–7.59)	0.218
Antiplatelet therapy	2.46	(0.299–20.3)	0.403
Both anticoagulation and antiplatelet	19.7	(1.20-322)	0.037
Non-steroidal anti-inflammatory agents	1.90	(0.942–3.81)	0.073
Clinical characteristics			
Time to antivenom >4 h	1.83	(0.861–3.88)	0.116
Thrombocytopenia	2.47	(1.24–4.93)	0.011
Platelet nadir	0.99	(0.988–0.997)	0.001
Fibrinogenemia	1.96	(1.01–3.81)	0.047
Fibrinogen concentration nadir	0.99	(0.993–1.00)	0.051
Increased international normalized ratio	2.67	(1.25–5.73)	0.011
International normalized ratio peak	1.13	(1.05–1.23)	0.002
Blister formation	0.52	(0.069–3.88)	0.521
Interventions			
Fasciotomy	6.14	(0.623–60.5)	0.120
Debridement	7.22	(2.18–23.9)	0.001
Antibiotics	1.78	(0.815–3.88)	0.148
Pain			
Early	1.07	(0.399–2.87)	0.893
Persistent	5.85	(1.03–33.2)	0.046
Early and persistent	2.35	(0.899–6.16)	0.081
Edema peak			
Thigh, bicep/tricep/deltoid	1.97	(0.854–4.54)	0.112
Groin, axilla/shoulder, torso	5.17	(2.27–11.8)	<0.001
Visible bleeding			
Inpatient	7.92	(2.35–26.7)	<0.001
Outpatient	0.94	(0.218–4.08)	0.938
Ecchymosis peak			
> half extremity	6.77	(3.39–13.5)	<0.001

Table 3. Univariate logistical regression for prediction of anemia, adjusted for age and sex.

Description	Odds ratio	95% confidence interval	*P* value
Bite Site			
Upper extremity	0.94	(0.453–1.94)	0.861
Home medications			
Anticoagulants	3.01	(0.812–11.1)	0.099
Antiplatelet therapy	5.50	(0.612–49.5)	0.128
Both anticoagulation and antiplatelet	55.4	(3.10-993)	0.006
Non-steroidal anti-inflammatory agents	2.59	(1.23–5.08)	0.013
Clinical characteristics			
Time to antivenom >4 h	1.82	(0.851–3.90)	0.123
Thrombocytopenia	3.03	(1.49–6.19)	0.002
Platelet nadir	0.99	(0.986–0.996)	<0.001
Fibrinogenemia	1.79	(0.909–3.51)	0.092
Fibrinogen concentration nadir	0.997	(0.993–1.00)	0.103
Increased international normalized ratio	2.52	(1.16–5.45)	0.019
International normalized ratio peak	1.12	(1.03–1.22)	0.007
Blister formation	0.60	(0.080–4.59)	0.626
Interventions			
Fasciotomy	7.40	(0.729–75.1)	0.091
Debridement	9.24	(2.65–32.2)	<0.001
Antibiotics	1.98	(0.896–4.38)	0.091
Pain			
Early	1.04	(0.384–2.79)	0.944
Persistent	5.94	(1.02–34.5)	0.047
Early and persistent	2.23	(0.846–5.89)	0.105
Edema peak			
Thigh, bicep/tricep/deltoid	2.11	(0.908–4.90)	0.083
Groin, axilla/shoulder, torso	5.86	(2.53–13.6)	<0.001
Visible bleeding			
Inpatient	10.1	(2.87–35.7)	<0.001
Outpatient	0.86	(0.198–3.75)	0.843
Ecchymosis peak			
> half extremity	6.17	(3.07–12.4)	<0.001

if enough blood loss occurs. However, patients generally must have significant derangements of clotting factors before they are at risk for spontaneous bleeding; for example, non-envenomated patients with thrombocytopenia are typically asymptomatic until the platelet count is $<50\times10^9$/L and are only at high risk of severe bleeding when the platelet count is $<10\times10^9$/L [12]. Further research is required to determine if these standard definitions are applicable in the setting of rattlesnake envenomation. Given that ecchymosis greater than half an extremity correlated with a four-fold increase in anemia, it is possible that the sequestration of erythrocytes under the dermis is significant enough to decrease hemoglobin values.

Erythrocyte destruction is another potential mechanism. While not commonly observed, a previous case report described intravascular hemolysis with Coombs-positive hemolytic anemia in a patient with North American rattlesnake envenomation without associated coagulopathy, implying a direct mechanism for destruction of erythrocytes [13]. Indirect destruction is also possible. Venom-induced consumptive coagulopathy is a pathology described in Australian elapids that is similar to but distinct from disseminated intravascular coagulation [14]. Australian elapid-induced consumptive coagulopathy causes microangiopathic hemolytic anemia due to widespread intravascular fibrin clot formation that mechanically destroys erythrocytes via shearing forces, resulting in schistocytes and diminished numbers of intact erythrocytes. Given the various etiologies for snakebite-associated anemia as stated above, further research should be done to quantify each to explore their relative impact on the development of anemia. Further research is also required before specific causal relationships can be determined.

In our study population, female sex was associated with more than a two-fold increase in anemia. This is unsurprising, given that female patients have lower hemoglobin values on average [15] and as a result

Table 4. Univariate logistical regression for prediction of anemia, type of antivenom administered.

Description	Odds ratio	95% confidence interval	*P* value
CroFab®			
Total vials	1.19	(1.09–1.31)	<0.001
Total vials, adjusted for age	0.99	(0.972–1.02)	0.655
Total vials, adjusted for sex	9.44	(2.85–43.2)	0.001
Anavip®			
Total vials	1.11	(1.06–1.17)	<0.001
Total vials, adjusted for age	0.99	(0.967–1.01)	0.279
Total vials, adjusted for sex	1.11	(0.386–3.04)	0.835

require a lesser degree of pathology to drop their hemoglobin below 100 g/L. When controlling for age and sex, patients with anemia were more likely to have concurrent derangements of platelets and INR values. Anemic patients were more likely to have severe coagulopathies, defined as a platelet count nadir ≤50 x 10^9/L [16], and a peak INR of ≥5.0 [12]. Anemic patients were also more likely to be on both anticoagulation and antiplatelet agents. It is unclear why dual anticoagulation/antiplatelet therapy is associated with anemia, and further research should be done to evaluate potential causes for this association. In our study population, anemic patients were administered more vials of antivenom. No specific objective measures have been established to indicate the need for further antivenom administration, but it is likely that the presence of anemia itself would cause a clinician to administer additional antivenom doses.

Anemic patients were more likely to have blood products transfused and had a lower recovery rate 14 days after hospital discharge. Given that anemia as a disease state can cause physical symptoms, it is possible that a patient's lingering anemia could contribute to the lower recovery rate. Transfusion of blood products in the setting of snake envenomation has not been investigated thoroughly, and the singular randomized control trial that used fresh frozen plasma in patients envenomated by Australian snakes showed benefit in the resolution of coagulopathy but no difference in time to discharge [17]. Further research should be done to investigate the role of blood transfusion in the setting of snake envenomation.

Our study has several limitations. Given the variety in snake venom internationally [18] and within the Arizona rattlesnake species [1], the generalizability of our results is limited. Additionally, since laboratory values are not continuously monitored, it is possible that some patients experienced undetected transient anemias. Baseline laboratory values were also not available for patients, and we are unable to verify that patients were not coagulopathic or anemic prior to envenomation. Our data set did not record the volume of crystalloid fluid a patient received, and we are unable to evaluate if hemodilution contributed to patient coagulopathies. Finally, our data are abstracted from poison center records retroactively and are subject to the biases inherent in these types of studies.

Conclusion

Anemia is a potential but uncommon complication in hemotoxic rattlesnake envenomations. In our patient population, anemic patients were more likely to be on chronic dual anticoagulant/antiplatelet or non-steroidal anti-inflammatory therapy. Anemic patients, when adjusting for age and sex, were more likely to present with thrombocytopenia and an increased INR. Anemic patients were significantly more likely to have ecchymosis extending past a joint space and to have widespread edema.

Author contributions

JD, SW, GS conceived and designed the study; JD, JK, AV, GS helped acquire and analyze data; all authors helped interpret data, and drafted, revised and approved this submitted manuscript, and are accountable for this work.

Disclosure statement

No potential conflict of interest was reported by the authors.

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Data availability statement

The data that support the findings of this study are available from the corresponding author, JD, upon reasonable request.

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