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ENVENOMATION BY THE MOJAVE RATTLESNAKE (*CROTALUS SCUTULATUS SCUTULATUS*) IN SOUTHERN ARIZONA, U.S.A.

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D. L. HARDY. Envenomation by the Mojave rattlesnake (*Crotalus scutulatus scutulatus*) in southern Arizona, U.S.A. *Toxicon* **21**, 111–118, 1983. Fifteen cases of envenomation by the Mojave rattlesnake (*Crotalus scutulatus scutulatus*) are reviewed. Systemic effects were observed in eight patients, consisting of early hypotension (3), decreased plasma fibrinogen (3) and platelets (2), elevated fibrinolytic split products (3) and eyelid ptosis (1). Local venom effects were most common and included swelling (15), ecchymosis (10), bleb formation (6) and necrosis (3). Effects upon neuromuscular transmission were neither common nor a clinical problem. Treatment consisted of i.v. crystalloid solution (15) and antivenin (12).

INTRODUCTION

OF THE MANY medically important pit vipers (*Crotalidae*) inhabiting the United States, the Mojave rattlesnake (*Crotalus scutulatus scutulatus* Kennicott) has been rated among the most dangerous (EMERY and RUSSELL, 1963; RUSSELL, 1969; PATTABHIRAMAN and RUSSELL, 1975; WINGERT and WAINSCHEL, 1975; RUSSELL, 1979; CASTILONIA *et al.*, 1981). This is based upon its venom's high lethality in mice (MINTON, 1959; EMERY and RUSSELL, 1963), blocking effect *in vivo* on neuromuscular transmission (RUSSELL *et al.*, 1961) and lethality in human beings (EMERY and RUSSELL, 1963; SMITH and RUSSELL, 1965; RUSSELL, 1979). However, there is relatively little documented clinical material available for the correlation with animal studies or comparison to effects in humans produced by other North American pit vipers (BOYS and SMITH, 1959; SMITH and RUSSELL, 1965; RHOTEN and GENNARO, 1968; MCFARLAND, 1973). This report presents the clinical course and medical management of fifteen patients with documented envenomation by *C.s.scutulatus* in southern Arizona. Some of the data contained in this report have been previously reported (HARDY, 1982a).

MATERIALS AND METHODS

Victims of envenomation by *C.s.scutulatus* were included in this study only when the snake involved was available to the author for identification and the patient was admitted to a hospital for treatment. In two patients, clear photographs of the dead specimens were accepted. The fifteen patients were cared for by different physicians in eight hospitals in Tucson, Phoenix and Wickenburg, Arizona. The author was consulted on and followed eleven patients first hand and consulted by telephone on three others. One additional patient was hospitalized during the author's absence but the frozen snake was available for identification and the patient was interviewed. The medical records were available for all patients. Legitimacy of the bite (KLAUBER, 1956; RUSSELL, 1980) was assigned as follows: a "legitimate" bite was inflicted when the danger of being bitten was not recognized, i.e., the snake was not seen beforehand, and an 'illegitimate" bite occurred despite the recognized risk, i.e., the snake was manipulated in some manner. Hematologic studies were by standard techniques in the Clinical Laboratories of the hospitals where the patients were cared for.

RESULTS

The data for fifteen patients envenomated by *C.s.scutulatus* are summarized in Tables 1 – 3. Ten patients were in or within 50 km of Tucson when bitten and five were in other areas of southern Arizona (Table 1). Although bites occurred throughout the year, 80% were in the June to October period. The high incidence of fourteen males to one female and twelve finger bites to three lower extremity bites (Table 2) is related to the high incidence of "illegitimate" bites (60%) (Table 1). This type of bite is unusual in females and specimens are more likely to be available for identification when the bite was "illegitimate". There was a predominance in boys of ages 4 – 11 years and young men of 19 – 31 years, and 67% were in or near their own dwelling when envenomated. Based upon length, seven of the specimens were newborn or juvenile animals and eight were adults.

TABLE 1. PATIENT PROFILE, HOSPITALIZATION AND BITE CIRCUMSTANCES

Patient	Date bitten	Locality	Sex	Age	City hospitalized	Hospital days	Activity when bitten	L—I*	Snake length(cm)
E.V.	12-12-75	Tucson	M	6	Tucson	4	Playing near home	L	41
T.M.	1-2-79	Tucson	M	22	Tucson	6	"Free handling" snake at home	I	102
A.H.	8-1-79	San Simon	M	4	Tucson	5	Walking barefoot near home	L	84
S.D.	10-12-79	48 km S. of Florence	M	11	Tucson	5	Walking in desert	L	82
R.S.	8-18-80	San Manuel	M	4	Tucson	2	Snake picked up near home †	L	23
K.C.	8-28-80	Marana	F	29	Tucson	1	Reaching into garden near home	L	41
R.D.	9-3-80	Tucson	M	19	Tucson	3	Picked up snake, impulse, desert	I	23
D.D.	9-4-80	Tucson	M	23	Tucson	5	Bagging snake at home	I	82
J.R.	9-9-80	Phoenix	M	20	Phoenix	2	Picked up snake, impulse, near home	I	30
C.R.	10-19-80	16 km S. of Tucson	M	31	Tucson	2	Picked up snake, impulse, desert road	I	82
D.E.	6-6-81	Gila Bend	M	28	Phoenix	4	Picked up snake, snake hunting, desert road	I	76
S.M.	6-8-81	8 km W. of Tucson	M	19	Tucson	4	Picked up snake, impulse, near home	I	41
J.J.	6-26-81	Tucson	M	23	Tucson	4	"Free handling" captive snake, at home	I	83
R.G.	7-24-81	Wickenburg	M	24	Wickenburg	2	Handling captive snake, motel	I	86
M.P.	3-31-82	Tucson	M	20	Tucson	5	Reaching under object in the desert	L	36

*L = "legitimate bite" = exposed to danger of being bitten unintentionally; I = "illegitimate bite" = unnecessary exposure to being bitten through rattlesnake manipulation.
† Danger not recognized.

The local effects following envenomation (Table 2) were characterized by swelling (100%), ecchymosis (67%), bleb formation (40%) and necrosis (20%). Systemic effects recorded were hypotension upon admission (20%) and abnormal hematological tests in 50% of those tested (Table 3). In the latter group, there were mildly decreased fibrinogen

TABLE 2. BITE CHARACTERISTICS, LOCAL AND SYSTEMIC EFFECTS

Patient	Bite location	Number of punctures	Local effects: Maximum swelling	Blebs	Ecchymosis	Necrosis	Systemic effects: Hypotension*	Neurological findings †
E.V.	great toe	one	to mid lower leg	no	no	no	no	no
T.M.	index finger	one	into thorax	yes	yes	no	no	no
A.H.	distal foot	two	foot and ankle	no	no	no	yes ‡	eyelid ptosis at 36 hr
S.D.	upper leg	two	into abdomen	no	yes	no	no	no
R.S.	index finger	one	into upper arm	no	yes	no	no	no
K.C.	middle finger	one	to elbow	no	yes	no	no	no
R.D.	index finger	one	to wrist	yes	yes	no	no	no
D.D.	index finger	two	into thorax	yes	yes	yes	no	no
J.R.	index finger	one	to mid forearm	no	no	no	no	no
C.R.	index finger	one	to upper arm	no	no	no	no	no
D.E.	index finger	one	to axilla	no	no	no	no	no
S.M.	index finger	two	to axilla	yes	yes	no	no	no
J.J.	index finger	two	to axilla	yes	yes	yes§	yes	no
R.G.	both thumbs	four	to elbows	yes	yes	yes	yes	no
M.P.	middle finger	one	to elbow	no	yes	no	no	no

*Hypotension: adult < 80 mm HG systolic; child (< 6 years) < 60 mm Hg systolic.

† Signs or symptoms of abnormality in neuromuscular transmission, e.g. diplopia, eyelid ptosis, dyphagia, dysphonia or respiratory muscle weakness.

‡ Creatine 1.8 mg/dl (normal 0.3 – 1.3) at 14 hr and 0.8 mg/dl at 42 hr.

§Amputation at metacarpal – phalangeal joint.

TABLE 3. HEMATOLOGICAL FINDINGS, ANTIVENIN ADMINISTRATION AND REACTIONS

Patient normal values	Anemia*	Fibrinogen † 150 – 300 mg/dl	FSP 2 – 10 µg/ml	Platelet † count 150 – 450 × $10^3/mm^3$	Antivenin vials i.v.‡	Time§	Serum reactions: Acute	Delayed
E.V.	no	138	N.D.	225	2	1.5	urticaria, facial edema, hypotension	unknown
T.M.	no	175	10 – 40	174	10	12	none	yes
A.H.	no	135	N.D.	381	10	4	none	yes
S.D.	yes Hb 9.8 Hct 27.9	350	N.D.	24	0		—	—
R.S.	no	190	N.D.	316	5	9	none	unknown
K.C.	no	200	N.D.	222	6	2	none	yes
R.D.	no	96	10	291	0		—	—
D.D.	no	300	10	270	5	1	none	none
J.R.	no	200	N.D.	223	5	2	none	unknown
C.R.	no	165	N.D.	226	10	2	none	unknown
D.E.	no	215	10 – 40	154	5	3	none	unknown
S.M.	no	170	10	187	"small amount"	1	urticaria, bradycardia, abdominal cramps, normotensive	unknown
J.J.	no	173	20 – 40	20	10	2	none	none
R.G.	no	N.D.	N.D.	N.D.	10	2	none	unknown
M.P.	no	N.D.	N.D.	157	10	2	none	yes

*Hemoglobin (Hb)<10 g/dl; hematocrit (Hct)<30%.

† Lowest value found.

‡ Wyeth Antivenin (Crotalidae) Polyvalent: one vial = 10 ml.

§Time antivenin was begun post-envenomation.

FSP Fibrin split products.

N.D. Not Determined.

levels (23%), elevated fibrinolytic split products (50%) and a decrease in the platelet count (14%). One patient (S.D.), with extensive ecchymosis of the lower extremity, had a significant decrease in hemoglobin and hematocrit five days post-envenomation. Eyelid ptosis was noted at 36 hr in a 4 year old male (A.H.), which lasted 18 hr. He also had only localized swelling without ecchymosis or bleb formation. Based upon local and systemic venom effects, all fifteen patients were judged by the author to have moderate to severe envenomation.

Treatment was primarily of i.v. crystalloid solution, e.g., lactated Ringer's injection and i.v. Antivenin (Crotalidae) Polyvalent, Wyeth. The thirteen patients given antivenin had negative skin tests to horse serum and eleven received an average dose of 7.8 vials without acute serum reaction (Table 3). Antivenin administration was begun an average of 3.4 hr post-envenomation. One patient (E.V.) received only two vials of antivenin because generalized urticaria and hypotension occurred, which responded to i.v. diphenhydramine and lactated Ringer's injection. The other patient (S.M.) had urticaria, abdominal cramps and bradycardia without hypotension after a "small amount" of antivenin. He responded to i.v. diphenhydramine, hydrocortisone, lactated Ringer's injection and s.c. epinephrine. Four patients had symptoms (primarily urticaria) of delayed serum reactions (serum sickness). Two did not have such symptoms and seven were lost to follow-up.

Residual venom effects were limited to areas of necrosis on digits. Primary healing occurred in one and amputation at the m-p joint was necessary in another because of periarticular loss of tissue around the proximal i-p joint. Digital contracture resulted in a third patient who refused further medical treatment. There were no contractures of extremities despite marked swelling in eight patients. Signs of systemic bleeding were not noted. Except for one patient who exhibited eyelid ptosis, none of the patients had other signs of venom effects on neuromuscular transmission, e.g. diplopia, dysphagia or respiratory muscle weakness.

DISCUSSION

Crotalus s. scutulatus is frequently confused with the western diamondback rattlesnake, *C. atrox*, in Arizona, southern New Mexico and western Texas (KLAUBER, 1930; GLOYD, 1940; JACOB, 1977). Its appearance is also similar to the Great Basin rattlesnake, *C. viridis lutosus* (AMARAL, 1929; KLAUBER, 1972). Such misidentification may account for the few clinical reports of venom effects following bites by *C.s.scutulatus*. The effects which have been described from clinical cases are as follows: local swelling (KLAUBER, 1956; SHANNON, 1957; BOYS and SMITH, 1959; RUSSELL, 1969; MCFARLAND, 1973), swelling of the entire extremity (RHOTEN and GENNARO, 1968), blebs, ecchymosis and necrosis (MCFARLAND, 1973), diplopia, dysphagia and dysphonia (KLAUBER, 1956; SHANNON, 1957), thrombocytopenia (RHOTEN and GENNARO, 1968), and circulatory – respiratory collapse in one patient resulting in death at 31 hr (SHANNON, 1965; SMITH and RUSSELL, 1965; RUSSELL, 1967; D. V. Porter, Safford, AZ, personal communication, 1978). Only one of the cases above was reported in detail (RHOTEN and GENNARO, 1968).

In contrast to these patient correlated reports, the venom of *C.s.scutulatus* has been characterized in the general literature as producing primarily neurotoxic effects in man (SHANNON, 1957, 1965; ARNOLD, 1970, 1975; PATTABHIRAMAN and RUSSELL, 1975; VAN MIEROP, 1976; WINGERT, 1980) and neurotropic effects in man and significant

effects on neuromuscular transmission which may predominate clinically (RUSSELL, 1962, 1967; RUSSELL and PUFFER, 1970; RUSSELL, 1971, 1973, 1975; RUSSELL *et al.*, 1975; WINGERT and WAINSCHEL, 1975; RUSSELL, 1977b; ANON, 1978; CASTILONIA *et al.*, 1979; WINGERT, 1980). It is stated that these effects may progress to respiratory muscle paralysis (SHANNON, 1957; RUSSELL, 1962; DOWLING *et al.*, 1971; RUSSELL and PUFFER, 1970; RUSSELL, 1973; WINGERT and WAINSCHEL, 1975; MINTON *et al.*, 1976; RUSSELL, 1977a; WINGERT and WAINSCHEL, 1977; ANON, 1978; WINGERT, 1980). In addition, local venom effects upon tissues are described as minimal (MINTON *et al.*, 1965; RUSSELL, 1967, 1969; RUSSELL and PUFFER, 1970; RUSSELL, 1975; MINTON *et al.*, 1976; WINGERT and WAINSCHEL, 1977; CASTILONIA *et al.*, 1979; RUSSELL, 1979; WINGERT, 1980; CASTILONIA *et al.*, 1981), i.e. little swelling or edema, pain, ecchymosis or necrosis (RUSSELL, 1962; EMERY and RUSSELL, 1963; SHANNON, 1965; RUSSELL, 1971; DOWLING *et al.*, 1971; PATTABHIRAMAN and RUSSELL, 1975; RUSSELL *et al.*, 1975; MINTON *et al.*, 1976; RUSSELL, 1977a,b; CASTILONIA *et al.*, 1979; RUSSELL, 1980; WINGERT, 1980; CASTILONIA *et al.*, 1981). *Crotalus s. scutulatus* has been compared to the tropical rattlesnake (*Crotalus durissus terrificus*) as having similar venom effects (RUSSELL, 1967; RUSSELL and PUFFER, 1970; RUSSELL, 1972, 1973).

The clinical effects of envenomation by *C. s. scutulatus* in the patients reported here were primarily in the local tissues and were similar to those reported for other North American *Crotalus* species (RUSSELL, 1979). Among these, local necrosis with digital contracture and amputation were the most significant. Of the systemic effects, early hypotension was dramatic in three patients and it responded promptly to i.v. challenge of crystalloid solution and i.v. antivenin. Hypotension later in the hospital course did not occur, most likely due to the judicious use of i.v. fluids. The venom of *C. s. scutulatus* has been shown to be fibrinolytic *in vitro* (DENSON *et al.*, 1972). Although venom effects upon laboratory tests for fibrinogen and platelets were observed, none of the patients had evidence of systemic bleeding. Eyelid ptosis occurred in one patient but other evidence of clinically significant venom effects on neuromuscular transmission were not observed.

The exact effect of antivenin administration cannot be defined, but the clinical course was without major systemic problems and the average hospital stay was less than four days despite the fact that moderate to severe envenomation was present in all patients.

A major, lethal protein from *C. s. scutulatus* venom was first isolated by BIEBER and TU (1974) and subsequently by HENDON (1975) and PATTABHIRAMAN and RUSSELL (1975). It has been designated Mojave toxin (BIEBER *et al.*, 1975) and *C. s. s.* Protein K′ (CASTILONIA *et al.*, 1981). This fraction has been found to have a pre-synaptic blocking effect on neuromuscular transmission in mice (CASTILONIA *et al.*, 1979, 1980; GOPALAKRISHNAKONE *et al.*, 1980; CASTILONIA *et al.*, 1981; HO and LEE, 1981) with death from respiratory muscle paralysis following rapid i.v. intoxication (GOPALAKRISHNAKONE *et al.*, 1980). Although BIEBER *et al.* (1975) found Mojave toxin to have cardiotoxic effects in rabbits, GOPALAKRISHNAKONE *et al.* (1980) showed no change in the rate or force of ventricular contraction in the rat heart. BIEBER *et al.* (1975) and HAWGOOD (1982) also noted a sudden fall in blood pressure following i.v. injection in rabbits. Local injection of this fraction in the mouse hind limb produces myonecrosis (GOPALAKRISHNAKONE *et al.*, 1980) which has been reported for other presynaptic snake neurotoxins (HARRIS *et al.*, 1980).

The local tissue and circulatory effects reported above appear to correlate with the clinical findings reported here. Effects on neuromuscular transmission, which are characteristic of Mojave toxin *in vitro*, however, were not seen clinically except for eyelid

ptosis in one patient. Clinical correlation is further complicated by the finding that *C. s. scutulatus* in south-central Arizona from Tucson to Phoenix lack this fraction in their venom and were designated "type B" specimens (STRAIGHT *et al.*, 1976; GLENN and STRAIGHT, 1978; GLENN *et al.*, 1983). The "type B" venom had proteolytic and hemorrhagic activity not seen with "type A" venom containing Mojave toxin (GLENN *et al.*, 1983). From a geographical standpoint, 12 patients in the present study were bitten by snakes from the "type B" locality. Of the others, one (A.H.) was in a "type A" locality and two (C.R. and R.G.) were in indeterminate localities. Investigation of additional clinical data and of venom from the specimens involved will be needed to determine whether the presence of the neurotoxic fraction is clinically significant. It is of interest that patient A.H. had eyelid ptosis. The possibility that the lack of clinically significant neurologic effects of the venom may be secondary to the use of antivenin cannot be ruled out. Since a number of fractions have been found in the venom (PATTABHIRAMAN and RUSSELL, 1975), further studies will be needed to determine their effects. Recently, HAWGOOD (1982) has reported *C. horridus atricaudatus* venom as producing blockade of neuromuscular transmission in the mouse phenic nerve – hemidiaphram preparation *in vitro* similar to that for *C. s. scutulatus*, whereas *C. h. horridus* venom had much less effect. Neurologic effects in human envenomation by *C. horridus* have not been reported.

Thirteen of the fifteen patients were hospitalized within the three year period 1979 – 1981, and ten of these were in Tucson. A recent study of *Crotalus* envenomation in Tucson (HARDY, 1982b) revealed that 159 patients were admitted with that diagnosis to eight Tucson hospitals over an eight year period (1973 – 1980). For the last five years of the study, the average was 24 admissions per year. The species involved was estimated to be *C. atrox* 50% and *C. s. scutulatus* 30%. There were no fatalities. RUSSELL and PICCHIONI (1977) estimated that 50 – 100 rattlesnake bites occur annually in Arizona.

Based on the clinical course of patients reported here, it appears that *C. s. scutulatus* in southern Arizona produces moderate to severe envenomation characterized by pronounced local effects, including necrosis. Hypotension following envenomation was the most serious systemic venom effect. Neuromuscular effects were not a major clinical problem. Treatment with intravenous crystalloid solution and antivenin appears to be appropriate. Clinical reports of human envenomation by specific snakes must include positive identification of each specimen involved.

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