

Ten years of snake bite in Madang Province, Papua New Guinea

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Abstract

A retrospective study was conducted of possible and definite snake bite admissions to Madang General Hospital, Papua New Guinea, for the years 1977 to 1986 inclusive. There were 175 such admissions and case notes were found for 129. Envenoming was recorded in 64 cases. Tender regional lymphadenopathy and ptosis were the most common signs of envenoming. No case of coagulopathy was recorded. 16 cases had clinical evidence of myotoxicity; in 3 of these a description of the offending snake suggested that it was the small-eyed snake (*Micropechis ikaheka*). Among 41 patients receiving antivenom, there was only one serious adverse reaction. This is the first study of snake bite in the New Guinea region; it shows similarities with, but also important differences from, snake bite studies in the Papua region of Papua New Guinea.

Introduction

Madang Province is one of 19 provinces in Papua New Guinea (PNG), situated north of the central mountain range in the New Guinea mainland region between 4° and 6°S.

Annual average rainfall is approximately 3500 mm, with the "dry" season extending from June to September inclusive. Population of the province at the 1980 census was 211 069. Most individuals are subsistence farmers living in rural villages. Madang General Hospital (MGH) is the major referral hospital for Madang town and the 27 rural health centres in the province.

There are 7 important species of venomous land snakes in PNG (Table 1), but only the death adder (*Acanthophis antarcticus*) and the small-eyed snake (*Micropechis ikaheka*) have been found in Madang Province. A retrospective study of snake bite cases admitted to MGH was undertaken primarily because published articles on snake bite in PNG, with one exception (BLASCO & HORNABROOK, 1972), all refer to snake bite in the Papua region which lies south of the central mountain range.

Materials and Methods

The admissions register for MGH was reviewed for the years 1977 to 1986. All admissions with possible or definite snake bite were listed. There were 175 such admissions and case notes were found for 129. Initial information sought from the notes included age, sex, time of the bite, part of the body bitten, circumstances of the bite, whether the snake was identified, and whether signs and symptoms of envenoming were recorded. There were 64 cases where envenoming was recorded; signs and symptoms were tabulated for

these envenomed cases. Types and amounts of antivenom used were also recorded, as were any adverse reactions mentioned in the case notes. One subset of 64 envenomed cases was selected for further analysis; it consisted of those patients with clinical evidence of myotoxic envenoming.

Results

Of 129 cases admitted with possible or definite snake bite, 111 were adults and 18 were children less than 12 years of age. There were 83 males and 46 females. There were 92 admissions during the wet season (October-May) and 37 during the dry season (June-September). The bitten limb was the leg in 105/129 (81%) and the arm in 10/129 (8%) cases; it was not recorded in 14/129 (11%) cases. 92 (71%) cases were bitten on the foot. The bite occurred during daylight hours in 80/129 (62%) cases and during the night in 42/129 (33%); it was not recorded in 7/129 (5%) cases. Analysis of the 64 envenomed cases revealed similar percentage results.

Tables 2 and 3 list the symptoms and signs (respectively) recorded in the 64 envenomed cases. In no case was there either any clinical evidence of coagulopathy or of prolongation of whole blood clotting time. There were 2 deaths, one of which was due to respiratory paralysis in presumed small-eyed snake envenoming. In the other case (snake not identified) death was probably due to respiratory paralysis although an adverse reaction to polyvalent antivenom could not be excluded.

There were 16 cases who showed clinical evidence of possible myotoxicity or rhabdomyolysis. All 16 had 2 or more of the following signs or symptoms: generalized muscle tenderness, neck stiffness, masseter pain, or dark urine. Of these, 10 cases also had neuromuscular paralysis; One was almost certainly a case of *Enhydryna schistosa* envenoming and has been reported elsewhere (HUDSON & FROMM, 1986). The mean duration from time of bite until admission was 35.6 h (range 1.5-72 h). A description of the snake was recorded in only 4 cases, in one as a "short black snake", but in the other 3 as a "long white snake". There was one death from respiratory paralysis. 5 cases had jaundice recorded, in 2 of whom urinalysis for bilirubin was recorded and was negative. Serum bilirubin was recorded in 2 cases and was elevated in both; however, both patients had received blood transfusions before collection of serum.

Dark urine was recorded in 11 of the 16 cases and, amongst these 11, 7 had generalized myalgia, 6 had a stiff neck or masseter pain, one had loin pain, and 3 had generalized muscle tenderness. In these 11 cases, mean time from bite until admission was 47.6 h (range 9.5-72 h). Urine microscopy was recorded in 3 cases, and erythrocytes were not seen in any. Testing for urine myoglobin or haemoglobin or serum creatine

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Table 1—Significant venomous land snake species of Papua New Guinea¹

Scientific name	Common name	Provinces ²
<i>Acanthophis antarcticus</i>	Death adder	W,G,C,MO,MD,ES, WS,EH,WH,CH
<i>Micropechis ikaheka</i>	Small-eyed snake	W,C,MB,N,MO,MD, ES,WS,EH,CH
<i>Oxyuranus scutellatus canni</i>	Papuan taipan	W,C,MB
<i>Pseudechis papuanus</i>	Papuan black snake	W,C
<i>P. australis</i>	King brown snake	W
<i>Pseudonaja textilis</i>	Eastern brown snake	MB,N
<i>Demansia atra</i>	Whip snake	W,C

¹Adapted from WHITAKER & WHITAKER (1982).

²C=Central; G=Gulf; W=Western; MB=Milne Bay; N=Northern; MO=Morobe; MD=Madang; ES=East Sepik; WS=West Sepik; EH=Eastern Highlands; WH=Western Highlands; CH=Chimbu.

Table 2—Symptoms recorded in 64 envenomed snake bite cases

Symptoms	Percentage of patients
In the bitten limb:	
Painful lymph nodes	59
Pain at bite site	36
Swelling at bite site	14
Local muscle pain	14
Systemic:	
Abdominal pain	56
Ptosis	55
Headache	33
Dysarthria	31
Dysphagia	31
Dizziness	28
Vomiting	25
Drowsiness	23
Diplopia	20
Generalized myalgia	14
Neck pain	9
Respiratory distress	9

Table 3—Signs recorded in 64 envenomed snake bite cases

Signs	Percentage of patients
In the bitten limb:	
Tender lymphadenopathy	52
Local swelling	19
Tender bite site	16
Local muscle tenderness	6
Systemic	
Ptosis	53
Bulbar paresis	17
Dark urine	17
Altered conscious state	14
External ophthalmoplegia	13
Generalized muscle tenderness	11
Stiff neck	7
Fever (>37.5°C)	6
Respiratory paralysis	5
Jaundice	5
Death	3

kinase was not available. Renal failure was documented in 6 cases who had dark urine. Mean blood urea was 299 mg% (normal range 5-40 mg%), with a range of 80-610 mg%. 2 cases required peritoneal dialysis.

Of the 64 envenomed patients, 23 did not receive any antivenom; none of these died. 3 cases had ptosis, which resolved without antivenom treatment. Of the 41 patients who did receive antivenom, 20 received polyvalent serum only, 18 received death adder serum only and 3 received both death adder and polyvalent sera. There were 10 adverse reactions to antivenom; 6 were to death adder antivenom and were manifested only as fever ($\geq 38^\circ\text{C}$) of less than 6 hours' duration. 4 adverse reactions to polyvalent antivenom occurred, of which 3 were febrile reactions only. The other case had an urticarial rash and bronchospasm which resolved after corticosteroid and antihistamine therapy.

Discussion

CAMPBELL (1964) analyzed data on 52 snake bite cases, admitted to Port Moresby General Hospital, from the Papua region of Papua New Guinea. The most obvious difference between the Papua and New Guinea regions of Papua New Guinea, concerning snake bite, is the distribution of venomous snakes (Table 1). In 3 of 5 New Guinea mainland provinces (East Sepik, West Sepik and Madang Provinces) there are only 2 significant venomous land snakes, the death adder and the small-eyed snake. Coagulopathy after snake bite in Papua New Guinea may be caused by the Papuan black snake, the Papuan taipan or possibly the eastern brown snake. Over a 10-year period at MGH there was no snake bite case with either clinically evident bleeding disturbance, or prolonged whole blood clotting time recorded. This is in accordance with herpetologists' findings on the distribution of snakes in Madang Province.

There are many similarities between Campbell's findings in Papua and those of our study. Snake bite occurred throughout the year in Madang also; most cases were bitten on the lower limb, especially on the toes and feet; and vomiting, headache, abdominal pain and local painful lymphadenopathy were common in both studies. Bilateral ptosis was found by Campbell to be the most common objective sign of

envenoming. Together with tender local lymphadenopathy, this was also the most common physical sign in our patients. Pain and swelling at the site of bite were uncommon in both studies.

50 of Campbell's 52 cases were bitten during daylight hours, and the other 2 were bitten before 1900h. 42 of our 129 cases (33%) were bitten at night, and the percentage was similar in the 64 envenomed cases. The death adder and small-eyed snake are quite active at night, whilst the Papuan black snake and Papuan taipan are more active during the day (SLATER, 1956). The presence of only the first 2 snakes in Madang Province probably accounts for the higher percentage of nocturnal bites.

Evidence of myotoxicity was not mentioned by Campbell. Dark urine was referred to in 3 of his 52 cases, but no mention was made of specific muscle symptoms. 2 of the cases with dark urine were reported in a previous study (CAMPBELL & YOUNG, 1961); one was haematuria and the other was considered to be haemoglobinuria. When coagulopathy is absent, dark urine after snake bite in Australia is more likely to be due to myoglobinuria than to haemoglobinuria (SUTHERLAND, 1983). In 3 of the 16 cases who had signs and symptoms suggestive of myotoxicity, the offending snake was described as a "long white snake". This is most likely to have been the small-eyed snake, as the only other venomous land snake in Madang Province, the death adder, never grows more than one metre in length. Furthermore, death adder venom has no myolytic activity (SUTHERLAND, 1983). This description of a "long white snake" is referred to in the only published article on small-eyed snake envenoming (BLASCO & HORNABROOK, 1972). Current knowledge on this snake has been reviewed recently (HUDSON, in press) and 2 cases of presumed small-eyed snake envenoming are described; both had generalized myalgia and muscle tenderness, dark urine, and severe neuromuscular paralysis.

Most of the 15 cases of land snake envenoming with myotoxic features presented late. Generalized muscle pain and tenderness, however, had their onset within the first 6 hours after the bite in some cases. The long delay between bite and admission, associated with rhabdomyolysis, was probably the main reason why acute renal failure developed in at least 6 of the 15 cases. It is highly likely that most, if not all, of these 15 cases were *M. ikaheka* envenoming. This would mean that *M. ikaheka* is responsible for a significant proportion of snake bite admissions in Madang Province. Clinical evidence of myotoxicity following a land snake bite, with or without neurotoxicity, would suggest *M. ikaheka* envenoming.

Other causes of dark urine in snake bite include haematuria, haemoglobinuria and, possibly, bilirubinuria. Concentrated urine may also be called "dark" by patients and misinterpreted by health workers. In a retrospective study, it is difficult to evaluate these possibilities. Haematuria is unlikely in view of the

failure to demonstrate erythrocytes in the urine in those cases who had urine microscopy. The common findings of muscle tenderness, pain and stiffness suggest that myoglobinuria was the likely cause of dark urine in the eleven cases in which it was recorded. Clearly, prospective snake bite studies in PNG, with estimations of serum creatine kinase and urine myoglobin and haemoglobin, need to be done.

CAMPBELL (1964) found that, of 39 cases given antivenom, 21 (53.8%) developed an adverse reaction, including urticaria (8 cases); fever (12 cases); cough (5 cases); bronchospasm (one case); and anaphylaxis (2 cases). In our study 10 (24.4%) of 41 cases receiving antivenom had an adverse reaction, a much lower proportion than that observed by Campbell but higher than the incidence of adverse reactions (12.9%) found in a study based on Australian and Papua New Guinean antivenom usage (SUTHERLAND & LOVERING, 1979). (Both Australia and PNG use the same polyvalent antivenom.) In our study, all reactions except one were febrile only. The single serious reaction occurred in a female patient who developed urticaria and bronchospasm whilst receiving polyvalent antivenom. She had not received any of the usual premedication (adrenaline and promethazine) and the infusion rate was too rapid. Whilst our study is retrospective, it indicates that antivenom used in PNG, when given correctly, has a low incidence of serious reactions.

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