Adder Bite in the British Army 1979-1988: A Decade of Experience

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SUMMARY: The results of hospital admissions for adder bite suffered by British Army personnel were reviewed over a ten year period. They confirmed the low bite rate and the risk factors previously demonstrated in earlier studies. In particular, the dangers of handling and attempting to catch the snake were highlighted. Also illustrated was a general ill-preparedness by clinicians to manage the condition. In this connection, the use of specific Zagreb anti-venom preparation for cases of severe poisoning is advocated, as is admission to hospital for assessment and appropriate monitoring. Adder bite is both unusual and unpredictable so caution needs to be exercised in the initial clinical appraisal.

Introduction

Of the three indigenous snakes in Britain only one is venomous. The adder, *Vipera berus*, possesses venom which it usually employs to subdue its prey¹. Its diet normally consists of small mammals, lizards, frogs and birds. These animals are killed by the effects of the venom which are generally described as being haemotoxic, cytotoxic and vasculotoxic². These are characteristics shared by many other venoms of the Viperidae³.

The adder is a snake which rarely grows larger than 60 cms. It may be distinguished by dark zig-zag markings along its back although they may be obscured if the background colour of the snake is itself dark. Thus, some individuals may be dark red or black. More usually, they are grey or brown. One feature, however, which tends to be constant is the prominent V shaped marking at the back of the head⁴. This may help identify the snake although the only reliable method is to examine the scale type and pattern. The adder's eye is elliptical with a typical viperid vertical slit, as opposed to the round eye shapes of the grass and smooth snakes¹.

Vipera berus is a wide ranging snake which is found throughout the United Kingdom and European continent from Northern Spain and Italy up to the northern regions of Scandinavia. In its more southerly habitats it overlaps with the northerly distribution of some other viperid snakes namely the aspic viper, *Vipera aspis*, the meadow viper, *Vipera corsinii*, and the sand viper, *Vipera ammodytes*⁴. The adder may be distinguished from the others by virtue of the inverted V shape at the back of its head and its preferred habitat. The snake prefers dry, sandy heathland and forest edges. Nevertheless, occasionally it may be found in wetter places. This is especially true when the weather is hot. At such times the reptile may withdraw to areas offering dampness and shelter⁵.

The adder exhibits marked seasonal variations in its habits. It goes into hibernation when the autumn temperatures begin to fall. This hibernation will last until spring, usually March, and the snake will resume feeding⁵. Mating takes place in April or May and the young are born in the summer. These timings are

approximate but are significant since they are clues to the activity levels of the snake. Thus, when emerging from hibernation the reptile will be sluggish but will possess a full load of venom⁵. During the mating season the snake is likely to be maximally active with a gradual slowing down during late summer. This pattern of activity is of importance in its effect upon human activities.

In common with nearly all snakes, the adder will make an attempt to avoid human contact⁷. However, if cornered or surprised the snake will attack. Its defensive posture is one of a loud hissing followed by a strike⁸. Notwithstanding this, many encounters with the adder are inadvertent. Equally, a proportion of adder bites are sustained whilst actually handling the snake⁵.

Method

This study is a retrospective survey of the reported cases held in the MEDSTATS programme. Clinical details were confirmed by reference to the hospital notes and results.

Results

Fig 1 gives the aggregate results of this series. There was a total of 13 patients admitted to hospitals after having sustained an adder bite. Clearly, the total only refers to actual documented hospital admissions. No data were available on those individuals who were bitten and did not warrant hospital admission. The figures refer to both civilian and military hospitals.

All the 13 cases were of male soldiers and only 2 of these were bitten accidentally; the rest were injured as a consequence of actually handling or trying to capture the snake. This is reflected in the site of bite, with 10 patients having suffered bites of the hands. Indeed, only one case in this series represents an attack on a part other than the upper limb; this was a bite on the right ankle.

The commonest month for adder bite was May with 5 bites out of a total of 13. June was the next most frequent with 4 bites, then July with 2 and March and September one each. Similarly, the location of the patient when bitten shows a gradation, with the commonest one being

PATIENT DETAILS	SITE OF BITE	LOCATION	TIME OF YEAR	PATIENT ACTIVITY AT TIME OF BITE	LENGTH OF HOSPITALI- SATION	SYMPTOMS	INVESTIGATIONS PERFORMED	ABNORMAL RESULTS	TREATMENT GIVEN	TIME TO COMPLETE RECOVERY
(A)	(B)	(C)	(D)	(E)	(F)	(G)	(H)	(I)	(J)	(K)
16 yrs M	R thumb	Northumbria	May	handling snake	2 days	local swelling	Fbc U+E CXR	nil	antibiotics	7 days
22 yrs M	R index finger	Surrey	June	handling snake	4 days	local and regional swelling, nausea	Fbc U+E ECG	nil	Piriton	15 days
18 yrs M	L forearm	Gloucestershire	July	trying to catch snake	4 days	local swelling	Fbc U+E ECG	nil	nil	10 days
26 yrs M	R forearm	Hampshire	May	accidentally disturbed snake	10 days	local and regional swelling, vomiting, nausea, hypotension	Fbc U+E CXR ECG Clotting studies	Moderate Leucocytosis in 4 days, T wave inversion in ECG	N/saline drip, intravenous hydrocortisone, antibiotics	29 days
19 yrs M	R hand	Wiltshire	May	handling snake	1 days	nil	nil	nil	nil	1 day
23 yrs M	L hand	Surrey	June	handling snake	8 days	local and regional swelling, nausea, vomiting	Fbc U+E CXR ECG Clotting studies	Moderate Leucocytosis in 4 days	antibiotics, intravenous hydrocortisone	21 days
25 yrs M	L thumb	Hohne, West Germany	March	handling snake	3 days	local swelling	nil	nil	antibiotics	3 days
22 yrs M	R middle finger	Hampshire	September	handling snake	5 days	local and regional swelling, hypotension, vomiting	Fbc U+E CXR ECG Clotting studies	T wave inversion in ECG	intravenous hydrocortisone, antibiotics	17 days
28 yrs M	L index finger	Herford, West Germany	June	handling snake	7 days	local swelling, necrosis later over bite area	Fbc U+E CXR ECG Clotting studies	Moderate Leucocytosis after 5 days	intravenous hydrocortisone, antibiotics, later surgical debridement and skin graft	82 days
35 yrs M	R index finger	Surrey	May	trying to catch snake	3 days	local swelling	Fbc U+E	nil	antibiotics	8 days
22 yrs M	R index finger	Hampshire	May	handling snake	2 days	local swelling	Fbc U+E	nil	nil	2 days
21 yrs M	R ankle	Wales	July	accidentally disturbed snake	2 days	local swelling	Fbc	nil	nil	4 days
20 yrs M	R middle finger	Yorkshire	June	handling snake	1 day	nil	Fbc U+E	nil	nil	1 day

Fig 1. British Army Adder Bites 1979-1988

the South East of England. Interestingly the total included 2 soldiers bitten whilst serving in Germany. The symptomatology showed the almost universal feature of local swelling. This is defined as a swelling of the afflicted structure and its immediate surroundings. For instance, local swelling of a bite to the thumb would include the thumb ball and the hand. Regional swelling would involve the whole of the upper limb.

In 3 cases significant nausea followed by vomiting was suffered. In 2 of these cases there was at least one episode of hypotension as well. Also of interest is the single case of necrosis over the actual area of snake bite. This is an unusual finding. Other abnormal results included 3 cases of moderate leucocytosis within 3 days of the bite. This was taken as a rise in white blood cell count of up to 17.3×10^9 per litre in the worst case. Similarly, there were 2 cases of T wave inversion in the ECG. Treatments given seemed to include mainly antibiotics, in 7 cases, and intravenous hydrocortisone, 4 cases. No treatment at all, other than reassurance, was offered in 5 cases. Specific anti-venom was not given at all in this series.

The recovery time to complete return to normal duties varied enormously. The average period of recovery was 15.38 days. However, if the complicated case of skin grafting onto the necrotic area is omitted, the average recovery time falls to 9.08 days. No cases of death or permanent disability were recorded in this series.

Discussion

Adder bite is a relatively unusual condition in England and Wales. It is naturally enough the result of adder incidence and human activity. In this context snakebite is a function of the interplay between snakes and human activities. This is a finding supported by Reid's work in Malaya⁹. Thus, the heathlands of southern England are particularly productive of adder bite given the relatively large populations of both adders and humans. Fig 2 gives the monthly bite rate. A striking feature is the absence of bites in August. This reflects the leave period of most army units and hence a decrease in the likelihood of soldier – adder interactions. Bites continue to be suffered by civilians as demonstrated in other studies. Within the United Kingdom, specifically the Aldershot area, Hawley described an average annual bite rate requiring hospitalisation of 3 per year⁷. Death from adder bite is fortunately rare. This series supports that finding. Between 1950 and 1972 in England and Wales, there was one reported death from adder envenomation but 61 from bee and wasp sting⁵. Similarly, long term disability from the venom is also uncommon. The one case which took 82 days to fully recover in this series, was subject to surgical intervention. It was the requirement to protect the skin graft that led to this long delay. Yet even including this long case, the average duration of recovery was 15.38 days.

Vipera berus venom is a complex mixture of proteases

whose combined effects are fundamentally cytotoxic and haemotoxic³. The poison is produced and stored in sacs which lie postero-inferior to the eye orbit. The venom is delivered to the victim by inserting the fangs into its tissues and pumping the straw coloured poison into the wound via the hollow tube of the fang⁶. The whole apparatus functions rather like a hypodermic syringe. Once delivered the venom acts by a variety of different mechanisms. The cytotoxic element seems to have an effect on vascular endothelial linings. This helps to explain the swelling seen in cases of envenomation. Indeed, early and massive oedema is a reliable indication of poisoning, swelling beginning as soon as 5 minutes after a bite has been described⁷. In any case, if no swelling has been seen 6 hours after a bite it is unlikely that significant envenomation has occurred^{5,10}. After 24 hours the swelling becomes overtly haemorrhagic. Again the damaged endothelial structure would explain this phenomenon. Another component of adder venom is an haemotoxic element¹². This certainly seems to be the mechanism of action in the preferred prey species. Nevertheless, in this series no evidence of coagulation abnormality was found. Similar findings have been described in other viperid envenomations³. The explanation may lie in the complexities of dose related responses. Adder venom has a much lower potency than many other viper poisons and is also delivered in small doses. Thus, in a large animal as represented by man, the small amount of venom may be insufficient to produce the systemic haemotoxic effects.

Another important element in adder venom is a direct cardiotoxic factor. This has not yet been isolated in adders but there is good evidence for its existence. Two cases in this series showed T wave inversion after envenomation. This relatively non-specific sign has been observed by others^{3,11}. An unusually severe cardiac complication was reported by Brown and Dewar¹³. They described a case of a 22 year old man who suffered myocardial damage, probably infarction, after an adder bite. A different problem of second degree heart block

Fig 2 Monthly Bite Rate

in a patient with a pre-existing history of ischaemic heart disease was described by Moore¹⁴. These more serious cardiac episodes are very much the exception to the general rule. The commonest manifestation of heart involvement in adder envenomation is T wave inversion. Confusingly, there seems to be little correlation between its presence and the severity of poisoning. Even mild cases of envenomation may show this trace⁵.

A much more sensitive indicator of severity of bite is white blood cell count. A moderate leucocytosis, specifically neutrophil, of between 16 and $26 \times 10^9/l$ was found in 3 cases in this series. These correspond to the most severely envenomed cases. Similar findings have been found elsewhere^{3,5,7}. Onset of leucocytosis may be as early as 2 hours after the bite but 24 to 36 hours seems more general. Certainly, any patient with early onset of swelling and neutrophil leucocytosis following adder bite must be assumed to have suffered significant and possibly severe envenomation. This is not to say that all adder bites lead to poisoning. Approximately 70% of viper bites actually cause envenomation⁹. Many factors affect whether a victim is poisoned or not. Amongst them are the time of year since emergence from hibernation is a period of maximal venom holdings. Similarly, time since last use of venom will also be of significance by virtue of venom store depletion¹⁵. Additionally, it seems that in many defensive bites, at least initially, the intention by the snake is to frighten and warn off rather than to envenom⁶. Clinical features of envenomation may well be confused in the early stages by the features of a fright response. The actual experience of suffering a snake bite is both physically painful and psychologically alarming. Nearly all patients exhibit some evidence of alarm and anxiety. These may cloud the rest of the clinical picture⁷.

Significantly in this series the commonest time for bites was May and June, with 9 incidents occurring in these 2 months. This is earlier than in other series⁵. However, in other respects this series confirmed others' experiences. Thus, all those bitten were male. A study in Sweden also found that men were more likely than women to suffer bites¹⁶. Other studies have also found this to be true^{5,9}. It seems that men are more likely to handle the reptiles. Interestingly, all but 2 of this army series were bites sustained whilst either trying to capture or physically handle the snake.

Having been admitted to hospital a variety of different treatments were given. The use of antibiotics was common, presumably on the basis that any bite was likely to be dirty and therefore infection a distinct possibility. Whilst this remains a theoretical risk there is no evidence that adder bite is an infective hazard. Nevertheless, antibiotic cover seems a prudent council. Anti-tetanus measures also seem to be advisable if existing cover has lapsed. Also of interest was that in 4 cases hydrocortisone was administered. Again, this was on presumptive grounds against toxic shock developing. The 4 patients who received the steroid were the most

seriously envenomed cases. Of real interest and indeed concern, was the omission of specific anti-venom from treatment regimes even of the most seriously poisoned patients. This may reflect the widespread confusion between the old Pasteur type anti-venom, which had real risks of anaphylactic reactions, and the more recent much safer Zagreb preparation¹⁸. Earlier uses of Pasteur solutions did indeed cause fatalities¹⁷. However, the definitive treatment for severe envenomation is Zagreb anti-venom¹⁸. This may be used even relatively late in the treatment; up to 4 days after the bite has been advocated⁹.

In the past some authorities have recommended killing the snake to aid in identification¹⁹. This advice seems to be misguided since in the United Kingdom and Northern Europe, the adder is the only venomous snake hence identification is not a real problem. The situation may well be different in the areas of the adder's distribution where other vipers are found. Even so, extreme care needs to be taken against further bites. It is worth noting that delayed head reactions have been described. In these, victims have been envenomed by a decapitated adder head up to 20 minutes after severance⁵.

Another piece of advice given, especially by American workers, is the incision and sucking of the site of the bite²⁰. There is no evidence that this is of benefit. Indeed, there are real dangers in this approach. Experience with adder bite suggests that this practice is at best ineffective and at worst positively harmful²¹.

Generally, there is no place in the acute stages for surgical intervention. The temptation exists to intervene when faced by the massive swelling of a severe envenomation. In such a clinical picture the pulses are often impalpable and so the question of surgical decompression may arise. Warrell advises against early surgical intervention and counsels objective assessment of blood flow before resort to surgery³. Access to doppler and other means of flow measurement is helpful. Even so a requirement for operative treatment in the early stages of adder envenomation is a rare occurrence. However, should the patient be subjected to surgery, recovery may be prolonged. Lau and Kenna described a case of fasciotomy performed on a 12 year old adder bite victim. His recovery was prolonged, taking 20 months, the implication being that the delayed recovery was due to the operative procedure rather than the residual effects of poisoning²².

Treatment Recommendations

Reid gave good guidelines for treatment of adder bite⁵. These include the following details. The first aid measures begin with wiping and covering the site but not definitively dressing it. Similarly, no attempt at either washing or incising and sucking the wound should be made. These practices may actually aid the onset of infection. The next stage of immediate aid is to immobilise the affected part if necessary by splintage.

This serves to delay the systemic distribution of the venom. Additional to this the importance of reassurance to the patient is paramount. Pain relief can be achieved by simple analgesics such as paracetamol. Seldom is any stronger pain relief necessary. Again, forceful and confident reassurance is most beneficial. Some advise that an absorption delaying bandage, rather than a tourniquet, should be applied. However, since the distinction between the two may not be apparent to the uninitiated the hazards of this device seem to outweigh its absorption delaying properties. Hence the recommendation is not to use them.

Timely resort to hospital is strongly advised. Snake bite in general, and adder bite is no exception, is an unpredictable condition. Hence, the wisdom of early referral and a full assessment by a hospital cannot be doubted. The hospital staff should reinforce any earlier reassurance and consider admission for monitoring. Reid believed that such admissions were much the wisest course because of the unpredictable nature of snake bite. Once admitted, the following need to be monitored:

1. vomiting, diarrhoea and abnormal bleeding.
2. hourly pulse, blood pressure and respiration.
3. white blood count, creatine phosphokinase and serum bicarbonate.
4. ECG (twice daily).
5. local swelling.
6. urinary protein and blood urea levels.

These indices will help to identify those suffering from moderate or severe envenomation. Symptomatic treatment and reassurance may be all that is required in cases of mild or even moderate poisoning. However, the definitive therapy for severe cases is the specific anti-venom. This is the Zagreb preparation and should be given intravenously. Indications for treatment are persistent hypotension, leucocytosis (especially over $20 \times 10^9/\text{litre}$), massive swelling, ECG changes (T wave inversion and/or ST segment depression) and raised serum creatine phosphokinase levels. Reid advised that the only role for steroids was in the management of delayed serum sickness reactions⁵. He also counselled caution in anti-venom administration with a known allergic history. Nevertheless, anti-venom remains the only definitive treatment. Interestingly, there are no paediatric variations in dosage. Administration should be intravenously, 2 ampoules in 100 mls of isotonic saline, using a rate of around 15 drops a minute⁵.

Conclusions

The adder is a species which seems to be getting more common²³. This may be a result of a succession of mild winters. At any rate any increase in the *Vipera berus* population is likely to lead to more envenomations. Certainly, the present low rate of bites seen in hospital represents only part of the overall picture of the

condition. Many victims probably do not bother to seek medical attention. This exact number is impossible to quantify. The condition is therefore unusual. Nevertheless, it is an affliction that needs to be taken seriously because of its very unpredictability. This small series has served to illustrate some of the problems encountered by clinicians involved in these cases. By virtue of its very infrequency, few doctors have the opportunity to build up a body of experience in the management and treatment of bitten patients. Thus, therapy tends to the symptomatic rather than the definitive. The lack of resort to specific anti-venom in this series, despite severe envenoming in at least 3 cases, serves to prove this. It is to be hoped that future cases of adder bite may benefit from the experiences of the clinicians involved in this series.

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ABSTRACTS OF PUBLICATIONS

MAJOR G E VINCENTI, RAMC

VINCENTI G E. Service families: a new perspective for the general practitioner. *Br J Gen Pract* 1990; **40**: 78-80.

Summary: "The psychological problems among the dependants of armed servicemen presenting to general practitioners are discussed, with particular emphasis on how the peculiarities of service life contribute to their aetiology and presentation. Within the UK, primary health care for service dependants is increasingly being provided by National Health Service general practitioners who have no knowledge of service life. An understanding of this minority group with its different lifestyle and subculture is important for management of these disorders".

MAJOR T O JEFFERSON, RAMC, G CAROLI, G MOLINARI and A P PEIRONE

JEFFERSON T O, CAROLI G, MOLINARI G and PEIRONE A P. Public Health and Community Medicine in England. *Rivista Italiana d'Igiene* 1989; **49**: 226-238.

Summary: This paper reviews the evolution of Public Health and Community Medicine in England. Their origins may be traced in a vast public movement which starting from the Victorian era aimed at the improvement of the sanitary and living conditions of the overcrowded and unhealthy slums of metropolitan England. The paper traces the evolution of the functions of the Medical Officer of Health (MOH) and the history of the discipline of Public Health through the two wars until the establishment of the NHS (1948), its first (1974) and second (1985) reorganizations. The origins of the Faculty of Community Medicine (1972), the decline of the specialty and its reorganization and renaming following the Acheson Report are also reviewed.

The historical review stops short of these proposed changes introduced by Working for Patients. These will be the subject of a second review.

MAJOR T O JEFFERSON, RAMC, H BRAND, E LEVRE' and G CAROLI

JEFFERSON T O, BRAND H, LEVRE' E and CAROLI G. The Personal Prevention of Malaria. *Rivista Italiana d'Igiene* 1988; **48**: 443-450.

Summary: Malaria represents a grave risk to the health of travellers to a country with malarial endemicity.

In order to diminish the risk of contracting the disease personal prevention measures coupled with the need to seek medical advice for any febrile episode are necessary.

The prevention of anopheline bites is based on simple personal protective measures such as the use of mosquito nets, insect repellent creams and lotions and insecticides together with chemoprophylaxis.

This has been made less efficient over the years by the appearance of *P Falciparum* strains which are resistant to several compounds and by the toxic effects of some antimalarial drugs.

In this paper the authors summarise three chemoprophylactic regimes currently advised in the United Kingdom to travellers to different parts of the globe characterised by different plasmodial resistance patterns.

Chemoprophylactic regimes recommended by other world health bodies are also mentioned.

Readers are also reminded that all patients with an appropriate travel history presenting with fever must be regarded at risk of malaria even if they have taken chemoprophylaxis.