Emergency treatment of adder bites: case reports and literature review

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SUMMARY

Five cases of adder bites are presented. The clinical effects and current recommendations for the treatment of adder bites are described based on a review of previously published literature. All adder bites, or snake bites of unknown species, should be observed for a minimum of 2h. Those with no local swelling may be allowed home with appropriate antitetanus prophylaxis. Patients with local reaction or signs of systemic envenoming should be admitted for 48h with ECG monitoring, and antivenom obtained. Indications and precautions for administration of antivenom are reviewed. Early antivenom treatment may reduce mortality and morbidity from adder bites.

INTRODUCTION

Vipera berus, the European adder, is Britain’s only native venomous snake and is distinguishable by a dorsal zigzag pattern and a V-shaped marking on the head. Adder bites are an infrequent cause of hospital attendance, although patients may fail to seek medical attention (Persson & Irestadt, 1981). There is a peak of adder bites in the summer months, usually in the upper limbs of males who interfere with the snake (Walker, 1945; Reid, 1976). The majority of patients are below the age of 15.

Only 14 cases of fatality had been recorded in the U.K. between 1876 and 1976 (Reid, 1976), but up to 50% of recorded cases have had significant local or systemic toxicity. A summary of clinical signs and symptoms with a classification of the severity of bite is summarized in Tables 1 and 2.

The clinical effects of adder bites are due to large molecular weight proteins.
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Table 1. Clinical features of adder bite

(1) Local (95%): oedema and haemorrhage spreading over 2–3 days, swelling persistent over 2–3 weeks. Regional lymphadenopathy.
(2) Gastrointestinal (24%): vomiting, abdominal colic, diarrhoea.
(3) Cardiovascular: hypotension (20%), sweating, ECG changes (T-wave flattening, arrhythmias 3%).
(4) Laboratory: leucocytosis (30%), raised CPK, thrombocytopenia (3%).
(5) Fever (5%).
(6) Depressed level of consciousness (5%).
(7) Pulmonary (3%) bronchospasm, pulmonary oedema, pulmonary haemorrhage.

Table 2. Classification of adder bites by severity (after Reid)

(A) Minor: no local swelling
(B) Mild: local swelling +/- gastrointestinal symptoms
(C) Moderate: extensive oedema +/- shock <2h
(D) Severe: shock >2h or other signs of systemic envenoming
(E) Fatal

which are taken up into lymphatics, initially, and then enter the systemic circulation. Hypovolaemia and local oedema is due to increased vascular permeability, at first due to the release of kinins, but later due to a direct effect of venom on the heart and blood vessels (Warrell, 1987). Local haemorrhage is probably due to cytolytic and haemolytic factors.

CASE REPORTS

Case 1

A 12-year-old boy was bitten on the finger by an adder in the Forest of Dean. There was no local reaction after 4 h. He was discharged with oral antibiotics after tetanus prophylaxis.

Case 2

A 10-year-old girl was bitten on her right middle finger by an adder in woodland. No local reaction was observed after 4 h. She was discharged on oral antibiotics.

Case 3

A 2-year-old boy was bitten on his right index finger in a Portsmouth garden. The dead adder was brought to the accident and emergency (A&E) department for identification. The child developed brusing and swelling of the finger and hand within 30 min of the bite. He was treated with oral antihistamines and steroids, and admitted for ECG monitoring, but had no systemic signs of envenoming over the following 48 h.
Case 4

A 13-year-old boy was bitten by an adder he was teasing outside a country pub. The dead snake was brought for identification. On arrival at the accident unit, some 25 min after the bite, he was pale, sweaty, confused and had a fine allergic rash over both upper arms. There was a 2 cm diameter area of bruising over the volar aspect of the right thumb with swelling of the hand and arm to just below the elbow. There was a sinus tachycardia of 130 min⁻¹ and blood pressure dropped from 110/70 to 80/50 mmHg. The patient was treated with 11 of gelofusine over 30 min, intravenous hydrocortisone and chlorpheniramine. Within 2 h of the bite he developed diarrhoea and vomiting and the oedema reached the chest wall. Results of routine biochemistry were normal, but coagulation studies showed a raised D dimer level >2500 ng.l⁻¹ (equivalent to increased FDP level), and there was a leucocytosis of 14.8 × 10⁹ l⁻¹.

Three hours after the bite 4.5 ml of Zagreb* antivenom was infused intravenously (the remainder of available antivenom had been used for Case 5). The patient then rapidly lost signs and symptoms of systemic envenoming. Residual haemorrhagic oedema of the right arm (Fig. 1) took 2 weeks to resolve.

Fig. 1. Residual haemorrhagic oedema of the right arm.

* Zagreb antivenom is supplied by Regent Lab, 861 Coronation Road, Park Royal, London NW10 7PT. Batches are tested by the Department of Health for quality before being placed in several pharmacies within each region. Hospitals should be able to obtain supplies at short notice.
Case 5

A previously fit 64-year-old man was bitten by an adder when he moved the snake out of the path of his car. He arrived in the accident unit some 30 min after the bite and 2 h after Case 4. He was accompanied by a live adder in a carrier bag. On examination he had a bite mark on his right hand with oedema of his hand and fingers spreading up the forearm. He was sweaty and hypotensive with a blood pressure of 95/60. ECG showed ST depression in V5 and V6. Routine biochemistry and haematology with clotting studies showed no abnormalities apart from a mild thrombocytopenia. The patient was treated initially with 200 mg hydrocortisone, 10 mg chlorpheniramine and 11 of gelofusine but his blood pressure failed to rise over the course of the following hour. He was then given 9 ml of Zagreb antivenom intravenously and became normotensive, pink and dry over the following 5 min. He then made a full recovery with the swelling slowly resolving over the following 14 days. He had a transitory itchy rash on day 7, possibly due to a mild form of serum sickness.

DISCUSSION

Cases 1, and 2 had no local reaction to the adder bite. The value of oral antibiotic prophylaxis after snake bites has been questioned (Persson & Irestedt, 1981), the majority of bacterial infections being due to unwarranted surgical interference. Splintage of the affected limb and antitetanus prophylaxis are the only local treatment required. If there is no local swelling after 2–4 h it is safe to assume that significant envenoming has not occurred (Warrell, 1987), and the patient may be discharged.

Case 3 illustrates a mild reaction to adder bite with local swelling but no signs of systemic toxicity. Since delayed hypotension and arrhythmias (Moore, 1988) have been described it is prudent to observe patients for 48 h, initially with cardiac monitoring. Investigations should include twice daily urea and electrolytes, full blood count, creatinine phosphokinase and clotting studies. The value of antihistamines and steroids is questionable. Antihistamines may help early allergic rashes (Case 4) and the sedative properties may be helpful in children. Most local and systemic effects are due to direct toxicity of adder venom, and the widespread use of steroids (including Cases 3–5) has not altered the natural history of reactions to adder bites (Persson & Irestedt, 1981). They should probably be reserved for treatment of late serum sickness (Reid, 1972).

Cases 4 and 5 illustrate severe reactions to adder bites with severe systemic signs and symptoms. Bringing a live or dead adder to A&E is not recommended, as second bites have been reported up to an hour after a snake head has been severed from the body (Reid, 1976). It can be assumed in this country that any immediate swelling from a snakebite or toxicity is due to the adder as this is Britain’s only venomous snake.

Administration of colloid is the mainstay of treatment of early hypotension; both Cases 4 and 5 were resuscitated with gelofusine. Antivenom was used in both cases, with marked clinical improvement. There is both experimental
Table 3. Antivenom treatment

<table>
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<th>Indications</th>
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<tbody>
<tr>
<td>(1) Hypotension persists &gt;2 hours despite IV fluids</td>
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<td>(2) Leucocytosis &gt;20 x 10^9</td>
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<td>(3) ECG changes</td>
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<td>(4) Acidosis</td>
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<td>(5) Raised CPK level</td>
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<td>(6) Local swelling in an adult seen within 2 h, or swelling at any age extending up the forearm or leg.</td>
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Contraindications

| (1) Hypersensitivity to horse serum.                                       |
| (2) Atopic/allergic history (relative contraindication).                   |

Dose

2 vials (9 ml) of Zagreb antivenom intravenously diluted in 100 ml 0.9% saline over 30–60 min. The antivenom dose is the same for all ages. Adrenaline should be available to treat possible reactions.

(Thakeston & Reid, 1976) and clinical (Cederholm & Lennmarken, 1987) evidence that early administration of antivenom reduces mortality and morbidity. Antivenom administered to envenomed Rhesus monkeys saved them from death and reduced subsequent local swelling. Local swelling may be prolonged in humans, especially adults, and early antivenom treatment may reduce severity of oedema and time to complete recovery. Until recently, antivenom treatment was unpopular in this country due to a single death in 1956 from anaphylaxis after administration of Pasteur antivenom. The newer, more active, and purer Zagreb antivenom is now sanctioned by the British National Formulary. Current indications and administration of Zagreb antivenom (Reid, 1976) are reviewed in Table 3.

Case 4 is unusual as clotting abnormalities have not previously been documented with adder bites, but are a common feature of envenoming by other vipers. The D dimer test is much more sensitive for fibrinolysis than the previous FDPs.

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REFERENCES


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