

Platypus Envenomation - A Painful Learning Experience

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Objective:

To describe in detail for the first time, the clinical course and medical management of a significant human envenomation by the Australian platypus (*Ornithorhynchus anatinus*).

Clinical features:

A 57-year-old man was envenomated via two spur wounds to the right hand from each hind leg of a male platypus. Pain was immediate, sustained, and devastating; traditional first aid analgesic methods were ineffective.

Intervention and outcome:

On admission to hospital, narcotics administered intravenously, both intermittently and by infusion, provided inadequate analgesia. A right wrist block was dramatically effective. After the blockade narcotic analgesic support was required for several days. The patient spent six days in hospital, and the envenomated area remained painful, swollen and with little movement for three weeks. Significant functional impairment of the hand persisted for three months, the cause of which is uncertain.

Conclusions:

Male platypus venom remains largely unstudied. It produces savage local pain and marked local swelling, but no apparent tissue ischaemia. No antivenom is available; in its absence the only effective analgesia appears to be regional nerve blockade, when the envenomation site and available skills permit. Immobilisation assists.

The Australian platypus (*Ornithorhynchus anatinus*) was first captured on the Hawkesbury River, New South Wales, in 1797 by David Collins.¹ A furry creature with a fat bill superficially like that of a duck, with webbed feet for swimming and a paddle-like tail, and claws on the webbed front feet used for digging burrows, it was viewed with disbelief when specimens were first seen by zoologists.

As well as its strange appearance it is a warm-blooded animal (mammal) - although the core temperature is maintained at only 33°C. Unlike other mammals, the female of the species (along with the Australian spiny anteater or echidna) lays eggs and suckles her young. Again, apart from the echidna, the male platypus is the only known male mammal which has "spur venom glands" - and the functional significance of the echidna's venom apparatus is doubtful.

The male platypus has a sturdy erectile keratin spur on each hind leg just above the webbed foot, which is connected via a venom duct to a venom (crural) gland lying under the dorsal thigh muscles.¹ The male envenomates the object of his aggression by erecting the spur, grasping and squeezing the victim (and simultaneously the crural glands) between his hind legs, and driving the spurs and venom into the victim's tissues. Under normal conditions this spurring appears to be a mechanism for opposing rival males during the mating season.

Platypuses are found in fresh water streams along the eastern Australian seaboard.¹ They survive equally well in warm Cooktown streams in northern Queensland and in icy-cold streams high in the Tasmanian mountains. A mature male platypus may weigh between 1.8 kg and 2.5 kg and reach 60 cm in length, with the tail being the final third. The female attains two-thirds the size of the male.

Platypuses are shy creatures that feed on insect larvae, small molluscs and small crustaceans on the bottom of their habitat streams. Feeding activities are usually around dawn and dusk. It has been demonstrated that the platypus (male and female) has electroreceptors in the bill, in addition to sensitive mechanoreceptors.' It may thus be able to locate and avoid inanimate objects by direct current signals, or locate and differentiate alternating current signals emanating from contracting muscles of some of its prey. This is the first such description in higher vertebrates.' More recently, similar electroreception capability has been demonstrated in the snout of the Australian echidna (*Tachyglossus aculeatus*).'

A platypus will usually dive quickly when approached and may stay underwater for up to five minutes. The animals live in burrows dug with their spade-like front claws in the sides of streams. The eggs are laid in special chambers at the end of the burrows and hatch 10 to 12 days later. The young are suckled via mammary slits or pores in the side of the female's abdomen, which exude milk; there are no teats. The young are weaned at about 16 weeks of age, maturity is reached by two and a half years, and they may live for as long as 23 years according to Fleay, who has for the first time successfully reared them in captivity.'

The male's hind leg spurs are strong and, when powerfully adducted, are able to support the animal's full weight while hanging from a larger victim. The spurs can be very difficult to disengage! The 2-4 ml of injected venom causes immediate and usually severe, disabling pain. It is not known if the venom's toxicity changes during the breeding season but a volume increase apparently occurs. The mating and breeding seasons are July to August in warmer areas, and October in colder southern areas. 2

No human fatalities or even near-fatalities from platypus envenomation have ever been reported. However, male platypuses have been described as spurring rival males to death, and similar deaths have been claimed in hunting dogs.'

Clinical record

A 57-year-old war veteran and Victoria Cross holder with previous experience of shrapnel wounds presented to the surgery of one of us (P J F) in May 1991 with intractable pain and marked swelling of his right hand.

Four hours previously he had been fishing at a mountain resort inland from Mackay, North Queensland, when he had seen a small platypus resting on a log in the middle of a river. Even when approached, the platypus did not move and he thought it might be "sick, or injured". He picked the platypus up by the back of the neck, in a similar way to holding a cat, and it moved very little. The platypus was then supported underneath and placed back in the water. Suddenly, in a wheeling motion one of the platypus' rear legs came back and struck the patient on the dorsum of his right hand causing the spur on the animal's hind leg (Figure 2) to drive into the flesh, angling towards the fingers. At the same time the spur on the other leg penetrated the radial side of the patient's right middle finger over the middle phalanx.

There was "immediate, severe pain". The platypus was difficult to disengage from the hand and had to be pulled in the reverse direction to enable detachment. Neither wound bled to any extent.

The pain and swelling in the right hand became worse, reaching peak intensity about one and a half hours after the original envenomation and not easing thereafter. The patient described the pain as "so bad I started to become incoherent". Even coughing caused a severe increase in the level of pain in the hand. The patient and his son tried heat treatment by soaking a towel in hot water and applying it directly to the wound, but found it of little benefit. Any movement of the hand greatly aggravated the pain. The envenomated area became hyperaesthetic; touching it, or even the weight of the hot towel, caused increased pain.

The pain was described as excruciating - much worse than previous shrapnel wounds the victim had suffered during the war. It was aggravated during the 100 km trip over rough roads to the doctor's surgery. A temporary arm sling elevating the hand gave little relief but prevented the pain from worsening with venous engorgement in the dependent position.

On arrival in the surgery four and a half hours after envenomation, the patient was in severe, unremitting pain. He had an obviously swollen right hand, with swelling most evident on his right middle finger. The hand colour appeared paler than usual but there was no ischaemia and capillary refill was normal. The right radial pulse was present and regular with a pulse rate of 120/min. His respiratory rate was 26 breaths per minute, and the blood pressure was 140/100 mmHg. On previous surgery attendances his blood pressure (seated) was recorded as 130/75 mmHg.

Intravenous access was obtained and 15 mg of morphine was given slowly. Over the next five minutes very little pain relief occurred. During this time we attempted to put his injured hand into hot (previously tested) water. However, the patient was unable to hang the hand down below heart level without further increase in the already unbearable pain.

An attempt was made to place the hand in a bag of ice while it was elevated. This too failed, as even small pieces of ice on the skin of the envenomated area greatly increased the pain. A further 7.5 mg of morphine was given intravenously 15 minutes after the first dose, but again this produced little relief. Over the ensuing 15 minutes a further 7.5 mg of morphine was injected intravenously, which eventually reduced the pain to a tolerable level. The patient remained alert with respiratory rate 24/min, pulse rate 100/min and blood pressure 140/90 mmHg. An electrocardiograph (ECG) taken at this time showed no abnormalities and no changes from one taken 3 years previously.

No attempt was made to slow absorption of the venom from the area by the use of compression / immobilisation techniques because of the absence of signs of systemic envenomation and the presence of the intense local pain and hyperaesthesia.

Tetanus prophylaxis was given before the patient was transferred to hospital, where he arrived at 11.30 a.m., five and a half hours after the envenomation. At this time his pulse rate was 74/min, blood pressure was 130/75 mmHg, and his temperature was 36.4°C. He was still in severe pain and so shortly after admission a morphine infusion of 1.5 mg per hour was commenced, followed by a full right wrist block with 20 ml of plain 0.5% bupivacaine. For the first time since envenomation the patient obtained significant pain relief. After swabs were taken from both spur wounds, the patient was given cephalothin sodium intravenously (1 g every six hours). The hand was elevated on two pillows.

Laboratory tests at this time included a full blood count and measurement of erythrocyte sedimentation rate (ESR), urea and electrolyte levels, renal function tests, liver function tests and levels of cardiac enzymes, with the creatine kinase level differentiated into cardiac and skeletal muscle fractions. These tests all gave normal results, except for ESR, which was 44 mm in one hour (normal range, 2-10 mm in one hour), total protein 56 g/L (normal range, 60-80 g/L) and serum albumin 26 g/L (normal range, 33-50 g/L).

On hourly observations the respiratory rate remained at 18-20/min but pulse oximetry showed the haemoglobin oxygen saturation (SpO_2) to be 87% breathing air. Oxygen administration via nasal prongs (2 l/min) was followed by an increase in SpO_2 to 97%. Vomiting and nausea commenced shortly after the morphine infusion was started but was controlled by a single intravenous injection of 10 mg metoclopramide. The hand remained numb and comfortable and so the morphine infusion was reduced to 1 mg/hour at 10.00 p.m. The patient was mildly pyrexical (37.5°C) but with no evidence of underlying infection.

Within two hours of reducing the morphine infusion the patient began to complain that the severe muscle and skin pain was moving "&I over". The pain "started moving from the right forearm into the upper arm, the right side of the chest and then it moved all over from the top of my head to the tips of my toes". It was worsened by any skin pressure; touching any area of the body made the pain at that site "severe". It was described as "feeling like a deep painful bruise". He also started to complain of a "swollen and sore throat but apart from slight reddening of the fauces, nothing was found. There was no right axillary or cervical lymphadenopathy.

The morphine infusion was again increased to 1.5 mg/hour, but although this appeared to reduce the right hand pain, it had little effect on the whole body symptoms. The wrist block was performed six hours after the envenomation and wore off some 18 hours later. Reasonable anaesthesia of the hand was then maintained with morphine infusion alone. No further wrist block was performed.

Twenty-four hours after the envenomation there was little change in the appearance of the right hand. The hand and fingers remained swollen but with a normal intact circulation. The right forearm gave the appearance of further swelling, but no initial measurements had been made. There was still no axillary lymphadenopathy. The patient complained of chest pain, so a precautionary ECG was repeated, which showed no change. Peak expiratory flow readings were normal at 400-450 l/min (predicted [Cotes), 520 Urnin)'l (the patient normally smoked 15 to 25 low nicotine cigarettes daily). Leg cramps responded to quinine sulphate (300 mg by mouth twice daily). An oral dose of ibuprofen (400 mg every eight hours) was commenced to see if it would help the muscle pains and hyperaesthesia. It did not, and as the patient was currently taking ranitidine (150 mg twice daily) for a previous duodenal ulcer, the ibuprofen was withdrawn.

Forty-eight hours after the envenomation the hand and forearm were still 'tender to touch', but were less hyperaesthetic. Ice and cold packs could now be placed on the area. The swelling was just as marked, but now finger movement was possible. Dextropropoxyphene napsylate by mouth (200 mg every four hours) was commenced. The morphine infusion was slowed to 1 mg/h and then ceased later that night, although a single 10 mg intramuscular injection was required during the early hours of the next morning, when the patient rolled on his injured hand in bed.

Three days after the envenomation, the swelling had decreased slightly and there was a little more movement in the fingers. The dorsum of the hand and the fingers were still very painful to touch, but the tenderness had moved down out of the forearm in a reverse sequence to its appearance; the "total body" pain had similarly subsided. Cold packs to the hand were now very effective as analgesics, but pain and tenderness returned as the area was permitted to warm. The pain was "tolerable".

Six days after admission the patient was discharged with his arm in a sling. There was 25% of the normal range of movement in the hand and fingers, except for the middle finger, which was still very swollen and had no movement. Twelve days later the patient had barely 10% flexion in this finger. He was still taking dextropropoxyphene and using cold packs every four hours for pain relief. There was sensitivity to touch of the right lower forearm, the dorsum of the hand, and all fingers. There was little forearm swelling. An oral dose of prednisone (12.5 mg twice a day) was commenced, and a review in a further three days was arranged.

At review, two weeks after the envenomation the patient still complained of pain and tenderness in the right hand. He was unable to put any pressure on the hand; he had difficulty cutting food, dressing himself, and signing cheques. The pain woke him at night if he compressed the hand. He felt that there was a "great improvement" since starting the

prednisone and that there was less "pressure pain" when using the hand. For the first time he was just able to hold the weight of a light jacket in his fingers. He still complained that he was unable to bear any weight (including a sheet in bed at night) on the back of his hand, as it irritated the hairs on the back of the hand causing a strange, painful sensation.

At this time significant forearm muscle wasting was observed. The whole arm was noticeably weaker, but precise strength testing (especially of the hand) was not possible because of pain and swelling. The circumference of the (dominant) forearm over the muscle compartment was 27.0 mm, compared with 285 mm at a comparable site on the left forearm. Similarly the circumference of the right upper arm was 290 mm compared with 310 mm on the left upper arm. The skin over the dorsum of the hand and the middle finger was now beginning to peel off. No localised hyperhidrosis or hypohidrosis or cyanosis was observed.

One month after envenomation the patient was still experiencing pain in the right hand when any muscular activity was attempted. He was unable to make a fist. Approximation of his thumb and fingertips was now possible, but flexion of his middle (envenomated) finger was less than 50%, and that of the ring finger was 60%. Other fingers had 100% flexion including the previously-deformed index finger. The forearm muscle mass had a smaller volume than two weeks previously (circumference now 220 mm), despite his being right handed. Upper arm measurements were unchanged.

Prednisone therapy was ceased after four weeks. Repeat blood tests showed a now-normal ESR of 10 mm in one hour but a leukocytosis of 14.6×10^9 (normal range, 3.9~10.6) with 77% neutrophilia. There were no clinical signs of infection, although it was difficult to exclude a tendonitis. The serum albumin level remained slightly low at 30 g/L.

Three months after envenomation the patient had diminished forearm muscle wasting but still had some stiffness and swelling of the middle finger and some swelling of the fourth finger. Hand usage steadily improved but was still not normal at four months after the envenomation. His right forearm measured 235 mm, his right middle and ring fingers and the dorsum of the hand still showed some swelling; making a full fist was still not possible.

When last examined, 19 weeks after the injury, the patient had a 10% reduction in hand clenching position, but normal strength of the interossei; bilateral forearm strength and dimensions were equal. The right middle finger was still a little swollen, with normal strength, but ached the day after being used.

DISCUSSION

Although 14 cases of envenomation by the platypus have been reported previously¹ none of these was described in close detail. A call to the local Poisons Information Centre and a further literature search were unrewarding. Despite this the Commonwealth Serum Laboratories "often receive calls about this subject" (Dr Struan Sutherland, personal communication).

General first aid principles applicable to the management of severe envenomation pain¹¹ .¹² were of no benefit for our patient. Only regional anaesthetic blockade, supplemented by narcotic intravenous infusion, administered in hospital served any useful purpose. (it is emphasised that these treatments should only be undertaken by trained persons, and in the presence of immediately available resuscitation skills and facilities.)

The patient himself noted that little bleeding occurred at the site of envenomation, although vigorous effort was necessary at the time to dislodge the platypus' spurs. The venom has been shown by pioneer researchers to have coagulant effects¹³. " but little else is known.

Although some limited fractionation studies were carried out by Temple-Smith in 1973³ the toxinology of platypus venom remains largely unstudied. The volume of venom probably increases near mating time, but it is not known whether any changes in toxicity occur. This envenomation occurred in May, which is approaching the northern Australian mating season for platypuses (July to August). However, the relevance of this in our case is unknown.

Corticosteroids given systemically have been advocated in envenomation, both for prophylaxis against serum sickness and as premedication before antivenom administration.¹ One of us (J W) has observed dramatic relief from previously unrelieved envenomation pain, 36 hours after a bite from an unidentified spider, with the use of systemic corticosteroids (intramuscular hydrocortisone, followed by oral prednisone).

Prednisone did not prove helpful for a previous platypus envenomation.¹ Our patient states that he felt very much better after its administration, with less pain and swelling. Such an effect may have been coincidental, or conceivably contributed to by the mood-modifying effects of steroids," although the onset was unusually rapid in our case. Corticosteroid-induced reduction in arm tissue oedema is another possibility, as is the reported benefit of steroids for reflex sympathetic dystrophy."

It is also not clear whether the muscle atrophy was a toxic effect of the venom on muscle and/or nerve tissue, an ischaemic effect from forearm compartment compression, a manifestation simply of disuse, or a combination of these factors? Not all the classical features of a reflex sympathetic dystrophy of the forearm and hand were present in our case, but they seldom are in any patient's. The profound, exquisitely triggered and spreading pain and hyperaesthesia, skin changes and proximal oedema were compatible with the diagnosis. (Apart from the initial wrist block, formal sympathetic blockade was not performed.) Further, an acute extensor tendonitis, a neuritis, or as mentioned, a compartment syndrome could not be confidently excluded, especially when symptoms and signs were at their worst.

The non-steroidal anti-inflammatory agent ibuprofen seemed to be of little benefit for our patient, although it was only used for 24 hours. Trial for a longer period would be possible in a patient without a previous history of duodenal ulcer.

It is hoped that the increasing forays of people into Australian bush do not threaten the platypus' existence, as it has done for other species. This is one of Earth's most remarkable animals. Clearly the male platypus venom can cause severe pain and morbidity. With a presumed one in two chance of picking up the wrong sex, perhaps the best advice for those venturing into the bush is "don't touch platypus". If such an animal really appears in need of assistance, the only safe way to handle it is by picking it up by the tip of its relatively long tail. The hind spurs thus cannot reach the "offending" hand. Handling the legs, head, or body, or attempting to wrap the animal in a cloth - no matter how thick - is fraught with the danger of being spurred.

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