

TOURNIQUET INJURY IN A PAPUAN SNAKEBITE VICTIM

Trevett A.J.(1), Nwokolo N.(2), Watters D.A.K.(2),

Lagani W.(4), Vince J.D.(5).

1. Wellcome Visiting Lecturer
2. Research Registrar
3. Professor of Surgery
4. Registrar in Paediatrics
5. Professor of Paediatrics

Dept. of Clinical Sciences, University of Papua New Guinea,
Port Moresby, Papua New Guinea.

Correspondence:

A.J.Trevett,

PO Box 2193,

Boroko,

N.C.D.

Papua New Guinea.

ABSTRACT

We report a case of snake bite complicated by tourniquet induced ischaemic damage and rhabdomyolysis causing acute renal failure. The case highlights the dangers of tourniquet use and of sudden release. We outline measures which can be taken to minimise morbidity in such patients.

KEY WORDS : SNAKE BITE, TOURNIQUET, RHABDOMYOLYSIS,
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CASE RECORD

A 9 year old boy was admitted to Port Moresby General Hospital (PMGH) on 23rd September 1992. He had been bitten on the right ankle by a snake 32 hours earlier at his village near Bereina in Central Province. At the time of the bite, his father had tied a tight grass tourniquet around his upper thigh. He had also made multiple razor cuts at the site of the bite. Two hours after the bite, the boy started to complain of pain in his right inguinal region. He subsequently vomited and developed a headache. He was seen at Bereina Health Centre 9 hours after the bite. At this time he was noted to have tender right inguinal lymphadenopathy, non clotting blood but no signs of neurotoxicity. The right limb was swollen, blue and pulseless. The tourniquet was removed and he was given a vial of polyvalent antivenom in saline, together with 25mg promethazine intravenously. He was also given tetanus toxoid.

Twenty six hours after the bite, he was noticed to be developing ptosis, dysarthria and progressive ophthalmoplegia. His right leg remained tender, swollen and pulseless. He was transferred to PMGH where he arrived 8 hours later. On admission he had complete ophthalmoplegia, ptosis and evidence of bulbar paralysis. His respiratory effort was poor and diaphragmatic. There was no muscle tenderness except in the ischaemic limb. His blood clotted

after seven minutes. It was impossible to localise the site of the bite due to the tissue swelling and extensive razor cuts around the lower leg. He was given a second vial of polyvalent antivenom, intubated and ventilated. He was oliguric and laboratory tests, not analysed until the following day, revealed marked renal impairment, hyperkalaemia and a grossly elevated creatine kinase level (Table 1). An ECG done at admission was unremarkable. He was given intravenous fluids and catheterised. 100mls of dark urine was obtained. The following day, he remained oliguric producing 200 mls of dark urine in 24 hours. He started to regain some eye movements. On the fourth hospital day, he was extubated. At this time he began to pass adequate urine volumes and the catheter was removed. His right leg remained swollen and tender but warm. Peripheral pulses were palpable. All eye movements were normal by day five.

Over the following ten days, the swelling in his right leg gradually diminished. He was unable to bear weight on the affected limb. He had limited power in his hip flexors and extensors, but no power in more distal muscles. He had no sensation below mid thigh at the level of the tourniquet. He was discharged and defaulted from follow up.

Table 1

	Adm	Day2	Day4	Day8	Day14
Na (mmol)	120	117	119	125	137
K	6.6	7.4	6.1	3.1	3.2
Cl	88	83	86	93	104
HCO3	19	16	18	19	22
Urea	26.2	40.4	41.1	26.8	4.2
Creat	150	300	360	200	80
PO4		1.81			
Ca		2.09(corr)			
CK	>5000 Units			3220	709
Myoglobinuria	+		-		
LDH	2475 Units			1710	

DISCUSSION

The case described above demonstrates three problems associated with the use of tourniquets. First, signs of envenoming appeared whilst the tourniquet was in situ, suggesting that it failed to prevent absorption of venom. Second, the child sustained considerable ischaemic damage to nerves and muscles in his leg and third, associated rhabdomyolysis resulted in acute renal failure and hyperkalaemia.

The use of tourniquets in the emergency treatment of snake bite has long been a contentious issue. In many rural areas of Papua New Guinea tourniquets are still routinely used. Often, as in this child, multiple vertical cuts are made around the site of the bite. Both of these practices may be a legacy from the then current advice given by kiaps during the Australian administration of Papua (1906-1975) although local incisions are traditional treatment in some areas (1). Whilst in some cases a tight tourniquet may slow or even prevent the absorption of venom it may also cause ischaemic damage and even death (2,3). Application of a tourniquet does not appear to be much more effective than a firm pressure bandage, which is designed to compress superficial lymphatics through which most of the venom is absorbed (4,5). In both circumstances the

compression should not be relieved quickly as there is a risk of large quantities of venom and toxic metabolites being released into the circulation with a concomitant sudden clinical deterioration (2). Release should be covered with antivenom, (although normal clinical indications for giving antivenom may be masked) and carried out where resuscitative facilities are available (6).

The "safe" period for a tourniquet which occludes arterial inflow is 1-2 hours. Once a tourniquet has been applied to a limb above arterial pressure for more than six hours, the risk of reperfusion injury due to the release of myoglobin, potassium and other toxic metabolites is so high that amputation without tourniquet release has been advocated as the best plan of management (7). Normally however, the severe pain produced by an arterial tourniquet prevents it being maintained in position for this period of time. In Papua, after an elapid bite, the progression of neurotoxicity to a degree requiring intubation is much longer than six hours in the majority of cases who have neither pressure bandage or tourniquet (Lalloo, Trevett unpublished). Only in patients in very remote areas could the slowing of absorption of venom conceivably save lives and if a tourniquet is used to achieve this the price may be loss of part or all of a limb. With other neurotoxic snakes in other parts of the world, in which the onset of neurotoxicity is faster, the situation may be different.

In the case described, the patient arrived in hospital almost 24 hours after the release of an 8 hour tourniquet. The presenting problem was one of a pale, pulseless immobile limb which was swollen but not tense. Fasciotomy would have been indicated had tension in the fascial compartments been causing or aggravating ischaemia and muscle damage. This was not thought to be the case, although no direct measurement of fascial compartment pressure was made. Clearly coagulopathy must be excluded or treated before considering a fasciotomy. Our "wait and see" policy was rewarded by the return of the foot pulses within 12 hours. With revascularisation, the limb did not become more swollen or tense so at no time did fasciotomy appear necessary. It must be conceded that tension within the fascial compartments cannot always be appreciated from without although the effects on distal pulses and capillary refill may be apparent. In a western society, amputation of a functionally useless lower leg and fitting of an above knee prosthesis may be the best method of rehabilitation. In the context of village life in the rural tropics however, amputation is a less satisfactory option and may be unacceptable in some societies.

The predominant biting species in the Papuan region is the Papuan Taipan (*Oxyuranus scutellatus canni*). The clinical syndrome of coagulopathy and progressive neurotoxicity exemplified by this case, is consistent with Taipan envenoming. Unfortunately, it was not possible to

establish serological proof of the biting species due to our inability to localise the bite site for a swab or aspirate, and because the boy had already received antivenom which was likely to make serum immunoassay useless. The venoms of both the Papuan Taipan (*Oxyuranus scutellatus canni*) (Lalloo, Trevett, unpublished) and the Australian taipan, (*Oxyuranus scutellatus*) (8,9), are mildly myotoxic. Although renal impairment is occasionally seen in patients bitten by Papuan taipans, we have not previously demonstrated it in association with myoglobinuria and comparable creatinine kinase levels. It seems likely that the myoglobinuria in this boy was secondary to tourniquet induced muscle injury rather than the myotoxic effect of the venom. Reperfusion of an ischaemic limb may generate the local production of free oxygen radicals causing further damage to myofibrils and myoglobinaemia(10). It is probable that other toxic metabolites released into his circulation from the ischaemic limb contributed to the deterioration in renal function.

The pathophysiology of acute renal failure in association with rhabdomyolysis is incompletely understood. A variety of hypotheses have been proposed, including direct toxic effects of myoglobin or its more toxic metabolites such as ferriheme (11). Dehydration and acidosis have been shown to be important factors in predicting the onset of renal failure in animal models (12). The majority of cases are seen in association with major trauma, but cases are also described after extreme exertion, overdose,

polymyositis, repeated seizures and hereditary metabolic causes such as myophosphorylase deficiency and carnitine palmitoyl phosphotransferase deficiency (13,14). The optimal treatment of patients with rhabdomyolysis includes alkaline diuresis in an effort to limit renal damage. One regime for traumatic rhabdomyolysis, suggests infusing the equivalent of 12 litres of fluid per day in a young 75kg adult, the suggested fluid containing 110mmol sodium, 70 mmol chloride, 40mmol bicarbonate and 10g mannitol in each litre of 5% dextrose (15). The benefits of this regime include controlling hyperkalaemia, correcting acidosis and dehydration, and preventing progression into established renal failure. This regime was not used in the patient described above. He presented long after the initial insult to the kidney and was already oliguric. A case could have been made however, for having diuresed him more aggressively and had his hyperkalaemia been detected earlier, more active measures should have been taken to correct it.

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