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Severe envenoming by a Gaboon viper (*Bitis gabonica*)

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A 42-year-old snake breeder summoned the ambulance after a fully-grown Gaboon viper (*Bitis gabonica*) had bitten him on the dorsum of his left hand. Fifteen minutes later the ambulance crew found the patient lying on the floor capable of communicating. An initial check showed blood pressure of 60/30 and a heart rate of 160/min. Fang marks were clearly visible on the dorsum of his left hand. Intravenous fluid and adrenalin administration were successful in stabilising the patient's condition to some extent. At the same time the local poison controller was sought via the local police. The pager in the patient's trouser pocket, however, indicated that he was actually the person sought. Meanwhile the patient's condition underwent generalised deterioration and he developed severe angioneurotic oedema without dyspnoea or stridor. He managed to advise medical staff where he kept the appropriate antivenom. While given adrenalin on a continuous basis the patient was taken to the local hospital as rapidly as possible.

On admission to hospital the patient appeared severely ill, with blood pressure of 80/40, heart rate 140, truncal erythrodermia, swelling of eyelids, lips and tongue, petechiae on tongue and palate, severe macrohaematuria and haematochezia. The laboratory findings were haemoglobin 20.3 g/dl; thrombocytes 11×10^9/l; INR 3.79; PTT 91 sec; fibrinogen 1.3 g/l.

Approximately one hour after the snakebite, the patient received 5 20-ml amp. antivenom (Saimr Polyvalent Snake Antivenom, South African Vaccine Producers). In view of his *unstable circulation* and a severe *coagulation disorder*, despite the supply of fresh frozen plasma, the patient was referred to a university hospital.

On admission a clearcut improvement in his condition was already noticeable and no clinical bleeding was apparent. The catecholamines could be withdrawn rapidly. Laboratory findings documented a favourable evolution 6 hours after infusion of the antivenom (haemoglobin 15.1 g/dl; thrombocytes 293×10^9/l; INR 1.1; PTT 27 sec; fibrinogen 2.27 g/l; d-dimer <0.5 mg/l [ELISA, Nycocard® Reader 2]; lactate 8.9 mmol/l; albumin 31 g/l).

Coagulation remained stable without an additional dose of antivenom. The left hand, however, showed progressive swelling with haemorrhagic blisters and progressive superficial necrosis on the dorsum. Observational treatment was initially chosen. Two days later, a severe compression syndrome of the carpal tunnel with entrapment of the median nerve developed, requiring acute relief

Figure 1
Gaboon viper (*Bitis gabonica*).

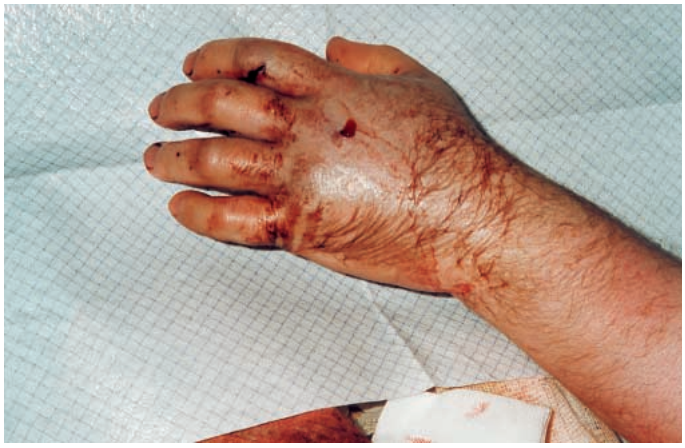

Figure 2
Progressive swelling and incipient necrosis on the dorsum of the left hand.

together with extensive debridement of the fang marks. Secondary healing and closure with skin grafts finally resulted in a uneventful local healing process and recovery of the full range of motion.

The Gaboon viper, *Bitis gabonica*, is a large viper widespread in sub-Saharan Africa. Despite its fearsome reputation, its extremely long fangs, and the large amount of venom it is able to produce, there are comparatively few recorded cases of bite in man [1]. In nature this snake is undoubtedly a docile animal, and the majority of reported bites have occurred from handling specimens in captivity.

Massive bleeding and hypotension are clinically the two most important features of envenomation by Gaboon viper venom (GVV). Bleeding is the consequence *of a disseminated intravascular coagulation (DIC)-like disorder* induced by gabonase, a thrombin-like enzyme, and other components interfering with the coagulation system. *In contrast to DIC only soluble fibrin is removed by the reticuloendothelial system and there is no formation of definitive fibrin clots, a fact which may explain why we measured normal fibrin degradation products.* In addition, the protease activity of haemorrhagins leads to disruption of capillary endothelial cells, and an antiplatelet compound has been isolated [1, 2]. Hypotension is promoted by vasodilatation, plasma leakage, and possibly cardiotoxic effects of GVV [2].

In this patient the very rapid onset of signs of envenomation is particularly remarkable. The early anaphylactoid reaction with generalising oedema and shock could be explained in different ways. It cannot be ruled out that the patient, who never experienced snakebite or antivenom treatment before, had been sensitised by handling and caring for this snake for several years. Autopharmacological properties of the venom inducing a massive discharge of histamine and other kinins were likely to promote the anaphylactoid reaction. Direct intravascular injection of venom could explain the rapid onset of bleeding within less than 30 minutes.

Massive local oedema and infection with clostridium spp (non-C. perfringens) may both have favoured the development of considerable necrosis. Although prophylactic antibiotic treatment after snakebite is not generally recommended [3], we feel that early administration of antibiotics – covering a broad gram-positive and gram-negative spectrum, including anaerobes – is probably indicated when a bite is located in sensitive tissues, e.g. the hand, and oedema occurs rapidly.

Late onset of coagulation disorders or recurrence after initially successful antivenom treatment have been reported [4], indicating the protracted release of venom from soft tissue compartments. In this patient the intravenous administration of 100 ml specific antivenom rapidly and definitively neutralised the systemic effects of the venom. The rapid normalisation of the coagulation parameters is indeed quite surprising, and we are unable to give a fully convincing explanation of this observation. If our hypothesis is correct, that the venom was directly injected into a vein, it could be argued that the haemorrhagic components were rapidly and completely inactivated by the antivenom – unlike in other conditions leading to DIC, e.g. septicaemia, where the cause of DIC persists for a longer period of time. We must also admit that GVV is a cocktail of many components with haemorrhagic and haemostatic activities whose interactions are not completely understood, and other reports have demonstrated that the development of coagulation disorders is highly unpredictable [1].

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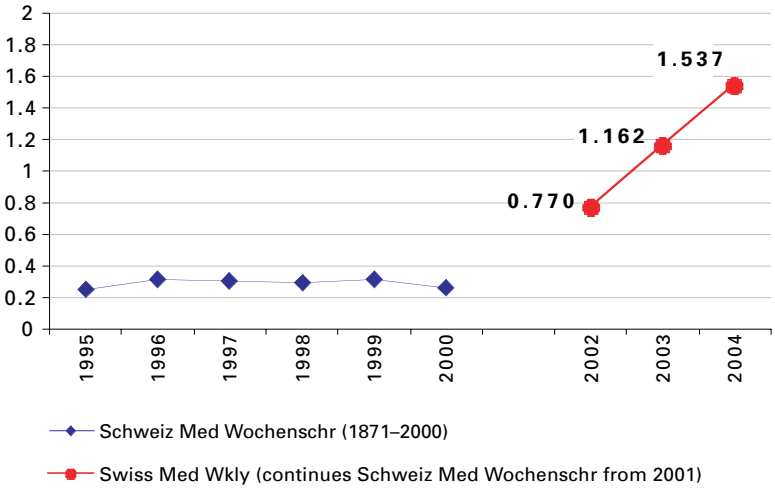

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