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Cerebrovascular accident after taipan bite

To the Editor: A 14-year-old boy was admitted to Port Moresby General Hospital (PMGH), Papua New Guinea, six hours after being bitten on the left ankle by a snake in Rigo, Central Province. He collapsed minutes after the bite and was semiconscious when seen at the local health centre one hour later. His blood was noted to be non-clotting at this time and he was given one vial of polyvalent antivenom by intravenous infusion and referred to PMGH. On arrival, he was semiconscious and irritable with a coma score of 8.[1] His blood did not clot and he was bleeding from gums, mouth and venepuncture sites. He had ptosis and reduction of eye movements. He was noted to have reduced power in his left arm. A bite site swab analysed with a Venom Detection Kit (CSL Limited, Parkville, Vic) was strongly positive for taipan venom.

Due to a shortage in the hospital, it was not possible to give the patient more antivenom. He was given four units of fresh frozen plasma after which his blood became coagulable. After progression of the neurotoxicity and involvement of both pharyngeal and respiratory musculature, he was intubated and subsequently ventilated for three days. His peripheral strength gradually improved but flaccid paralysis persisted in his left arm and he was noted to have left facial weakness. Later, hemisensory loss affecting the left arm and dyspraxia were found, but there was no apparent visual field defect. The patient absconded on the tenth hospital day, by which time he had regained some power on the left but still had a marked functional deficit.

The venom of the Papuan taipan (*Oxyuranus scutellatus canni*) contains procoagulants and haemorrhagins. Abnormalities of haemostasis are a cardinal sign of envenoming, and bleeding from cuts, venepuncture sites and gums is common. This patient's signs were consistent with a spontaneous bleed in the territory of the right middle cerebral artery, possibly in association with a predisposing vascular abnormality. Intracranial bleeds have been reported following the bites of other elapid snakes[2-4] and occur occasionally in patients bitten by Papuan taipans (Lalloo & Trevett, unpublished observations). It is interesting to note that this vascular event occurred within 30 minutes of envenoming, before the administration of antivenom. The mechanism could have been intracranial thrombosis occurring during the early, hypercoagulable phase of envenoming. There is a theoretical risk that giving a patient clotting factors before circulating procoagulants have been adequately neutralised by antivenom could exacerbate this problem.

Early collapse is not uncommon in victims of taipan bite. The mechanism remains obscure in the majority of cases and most are not associated with persisting neurological deficit. As has been described in patients envenomed by several other elapids, the coagulation abnormalities produced by the venom of the Papuan taipan can cause cerebrovascular accidents. It is possible that this may be a mechanism of some of the sudden early deaths which occur in taipan bite.

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In reply: It is most unfortunate that it was not possible to give this patient more antivenom "due to a shortage" in the Port Moresby General Hospital. If taipan antivenom was not available, polyvalent antivenom would have done. If either antivenom was there, and not used, the patient had good reason to abscond on the tenth hospital day.

There is no mention of first aid in this case. Since the pressure immobilisation type of first aid has been actively promoted in Papua New Guinea by various organisations, including Rotary International, it seems a shame it was not used. On the other hand, it is pleasing that a CSL Venom Detection Kit (CSL Limited, Parkville, Vic) confirmed the diagnosis that a taipan was involved, although this investigation did not benefit the patient in the least.

Intracranial bleeds after snakebite in Australia are extremely rare. Five have been reported this century, and three of these were associated with the intravenous use of adrenaline. At CSL we have no records of intracranial haemorrhage after bites by taipans in Australia.

There is more than a theoretical risk of embolism and thrombosis if clotting factors are given to the patient prior to antivenom therapy, or when antivenom therapy is markedly inadequate. Dreadful complications have occurred in the past, particularly when patients have received whole blood prior to antivenom therapy.

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Congenital syphilis: when the medium fails to transmit the message

To the Editor: Gurry and colleagues[1] have raised important issues in their report of two cases of congenital syphilis. They suggested that the complexity of serological reports for syphilis contributed to the failure to detect maternal syphilis and concluded that such reports would be more helpful if they were issued with an interpretation.

While I agree that there is a need to optimise communication I feel they are being unrealistic in laying the blame on the laboratory reports. The suggestion that the requesting doctor be supplied with the "correct" interpretation implies that an accurate clinical opinion can be formed from the serological result alone. This is not the case. The evaluation of a pregnant patient whose serological test results are positive for syphilis must include a history and physical examination,[2] plus knowledge of previous serological status. Doctors issuing serology reports without full clinical information have limited scope for interpretation. Comments made on reports can explain and expand the information presented on the report but a clinical consultation would be required to establish appropriate management. Doctors receiving serological reports for syphilis should seek expert advice if unsure of the appropriate clinical response. "Shooting the messenger" will not help.

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In reply: The letter from Dr Lyon reflects a response similar to that which we have experienced in direct communication. Clinicians in many disciplines have spoken warmly of our "cautionary tale", but some laboratory personnel have taken it initially as an unfair criticism of them. This is upsetting, but we have been able to persuade laboratory colleagues that we are being fair in describing a problem in the transfer of communication, that this problem is real, and that there is no value in territorial protection.

We didn't lay the blame on the laboratory reports — we wrote that a factor in the problem was the lack of appreciation of the significance of the reports. In the evaluation of the first case there was no history and no signs on examination, other than the pregnancy. In the second case there was incomplete background information about syphilis. However, in both cases the cardinal information indicating syphilis was serological. There was a lack of appreciation of this by those managing the pregnancies.

We hoped to aid understanding between clinician and laboratory by noting "there are valid