the literature that exists, remains uncertain. The behavior of MM and its subtypes has yet to be defined and requires extended patient follow up, with larger patient series. Our patient is being followed very carefully with regular scheduled clinical and radiological follow-up

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To the Editor

Fatal Cerebellar Hemorrhage Following Australian Brown Snake Envenomation

Venomous snakebites cause significant global morbidity and mortality, but their sequela are rarely encountered in neuropathology practice in North America. This case illustrates the potential dire consequences of a bite from one of the world's most venomous snakes, the Australian brown snake.

A 69-year-old female with a past medical history of hypertension stepped on an Australian brown snake outside of her home in Western Australia and was bitten on the instep of her foot. Appropriate pressure-immobilization first aid was initiated and the Emergency room assessment within 15 minutes of the bite revealed an INR of 0.9 and a baseline Creatinine of 191. Thirty minutes after the bite she began feeling unwell, vomited, started bleeding from her IV site, and her INR was 10. Brown snake venom was detected in her urine and she was treated with 5 Units of Brown snake anti-venom. Over the course of the next 48 hours her coagulopathy worsened (INR > 30), she was persistently hypertensive (up to 235/150 despite medical management), and she developed microangiopathic hemolytic anemia and renal failure (maximum Creatinine of 303). When her level of consciousness deteriorated a computed tomography (CT) scan showed an intraparenchymal hemorrhage in the posterior fossa (Figure 1). She died within 48 hours of being bitten.

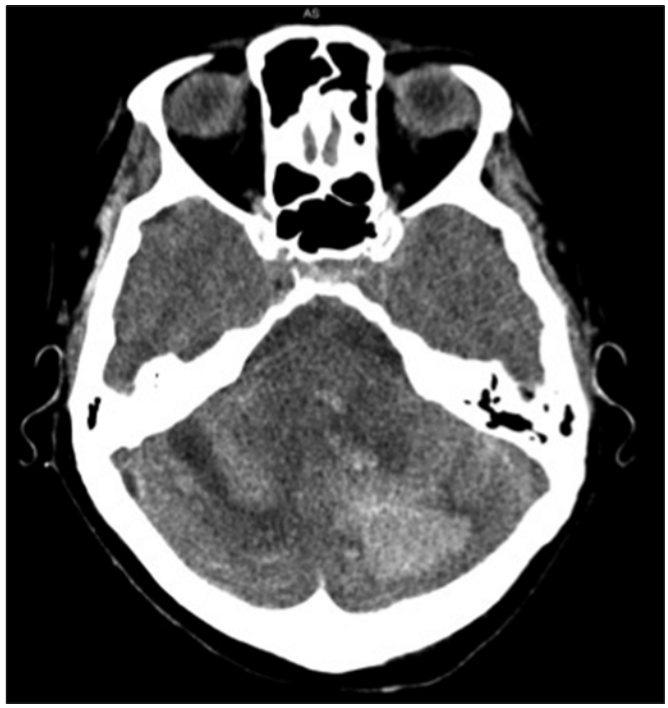

Figure 1: *Axial CT scan showing intraparenchymal hemorrhage within the cerebellum.*

At autopsy the brain was swollen with tonsillar herniation and a 50 x 25 mm V-shaped hemorrhage within the cerebellum (Figure 2). Histologic examination showed no evidence of an underlying vascular malformation, neoplasm or infarct.

Venomous snakebites are a significant global health problem, with an estimated five million cases of snake envenomation resulting in 125,000 deaths worldwide each year[1]. Many of these fatal envenomations are from *Naja* species (cobras) in Asia, or from Viperidae species in Africa[1]. In the United States there are 7000-8000 snakebites resulting in five or six deaths a year[2], mostly from Rattlesnakes. There are four species of venomous snakes in Canada but reports of significant morbidity and mortality from snakebites in Canada are few. The Australian Snake Bite Project has documented between 1000-3000 snakebites and four resulting deaths a year within Australia. All of the Australian deaths have been due to one of three snake species: Australian brown snakes (most commonly), tiger snakes

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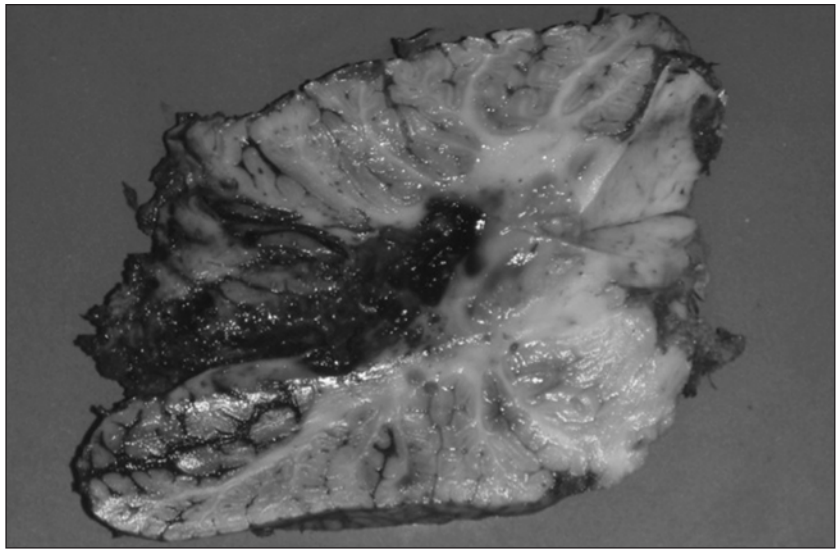

Figure 2: *Parasagittal sectioning through the cerebellum demonstrates a V shaped intraparenchymal hemorrhage.*

and taipins, all of which are members of the Elapidae family and are among the most venomous snakes in the world. In developing nations snakebites are an occupational hazard among workers in agricultural plantations and have a higher case fatality rate due to the scarcity of anti-venom and health care resources, while in developed nations snakebites are a recreational hazard, with a notable number of bites occurring during intentional handling of the animal.

Venom from a single snake contains many different enzymes with a myriad of potential resulting clinical effects. Some venoms result in cytolysis manifesting as marked edema and necrosis at the bite site, a frequent feature of Diamondback Rattlesnake bites in North America. Sea snake venom can contain pre or post-synaptic neurotoxins. The varied hemotoxic effects of snake venom include coagulopathy, such as the coagulopathy seen in victims of the Diamondback Rattlesnake, responsible for 95% of snakebite deaths in the United States. Half of patients bitten by this snake develop defibrination syndrome due to the presence of a 'thrombin-like-enzyme', *crotalase*, in the venom of this species[3].

Thirty-four cases of Australian brown snake envenomation were reported to the Australian Snake Bite Project between 2002-2006. The usual clinical consequence of envenomation by this species is Venom Induced Consumptive Coagulopathy (VICC). A potent *pro-thrombin activator* in the venom causes VICC by converting fibrinogen to fibrin, causing a brief initial pro-coagulant phase (with rare causes of sudden cardiac death from massive coronary thrombosis) followed by an exhaustion of fibrinogen and subsequent clotting defect. With early anti-venom treatment the pro-thrombin activator can be neutralized with normalization of clotting function over 6-18 hours. Four of the 34 reported cases also developed a thrombotic micro-angiopathy, similar to our case, with thrombocytopenia, microangiopathic hemolytic anemia and acute renal failure. This phenomenon is of uncertain pathogenesis, and delay to anti-venom treatment can be a contributing factor[4]. Within the 1990's there were four reported fatal intra-cerebral haemorrhages from Australian brown snake envenomation with hypertension as a known confounding factor[5].

The mainstay of treatment for Australian snakebites is pressure-immobilization first aid and anti-venom therapy, which is initiated at the first clinical symptoms of envenomation as hypersensitivity reactions to anti-venom are a significant problem.

In summary, this case demonstrates that the potential hemotoxic effects of Australian brown snake venom include VICC and intra-cerebral haemorrhage, which can occur despite appropriate and timely medical management. The patient's hypertension was likely a confounding factor.

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