Hump-nosed pit vipers and their bites in Sri Lanka

*Hypnale hypnale* (Merrem, 1820), *H. nepa* (Laurenti, 1768) and *H. zara* (Gray, 1849)

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**Introduction**

The hump-nosed pit viper (*Hypnale hypnale*, Merrem’s pit viper) is a snake widely distributed in Sri Lanka and the South Western coastal region of India. It is the commonest snake responsible for venomous snakebite in Sri Lanka, estimated to be between 22 to 77% of all snakebites (de Silva, 1981; Seneviratne, 2000). For centuries it was considered to be a relatively innocuous snake till 1821, for the first time, swelling and bleeding due to bites by *H. hypnale* was reported in animals (Davy, 1821).

Since then, apart from a few isolated case reports of renal injury and death, very little was published about the clinical features following its bites and the morbidity and mortality that resulted. *H. hypnale*, Merrem’s pit viper, was the species recognised to cause morbidity. However, clear detailed information about other species of hump-nosed vipers, and their clinical, toxinological and biological properties were not well studied nor documented till the turn of the twentieth century. Thereafter, many accurately documented papers about hump-nosed viper envenoming have appeared in the published literature.

The hump-nosed viper is widely distributed in all the penepalanes of the country and is commonly found in coconut, rubber and tea plantations. Three species of hump-nosed vipers of the genus *Hypnale* are found in Sri Lanka. *H. hypnale* is widely distributed except in Jaffna. *H. nepa* is confined to the central hills and *H. zara* to the lowland rain forests of the south-western wet-zone and the foothills of the central highlands. All are venomous and look alike superficially, being different as regards scale counts. *H. nepa* and *H. zara* are endemic to Sri Lanka while *H. hypnale* occurs in the Western Ghats of peninsular India as well (Maduwage et al, 2009).

**Epidemiology**

Recent studies have focused on identifying possible differences between envenoming caused by the three species. There does not appear to be a difference shown by results published so far; larger series are needed to show up any possible variations (Maduwage et al, 2013; Rathnayaka et al, 2017b).

The majority of bites occur in the evening hours. As a result of the short striking distance most bites are seen in the extremities—the fingers, toes and feet, below the ankles. In the study by Maduwage et al (2013) of 114 bites (93 *H. hypnale*, 16 *H. zara* and 5 *H. nepa*) most were on the lower limbs and had occurred in the daytime.

**Toxinology**

The venom of all three species is essentially similar with potent cytotoxicity, mild anticoagulant, pro coagulant and haemolytic activity. It also has weak myotoxic, neurotoxic and nephrotoxic effects. Most clinical effects are due to phospholipase A2 activity (Maduwage et al, 2011b). The same workers showed that Indian polyvalent antivenom raised...
against *Naja naja*, *Daboia russiellii*, *Bungarus caeruleus* and *Echis carinatus* did not neutralize the venom effects, consistent with what the previous studies of Tan et al (2011) had shown. Sellahewa et al (1995) had shown that polyvalent antivenom was not effective in treating severe local envenoming caused by hump-nosed viper bite.

**Clinical features of envenoming**

The most consistent effects of envenoming are severe local pain and local swelling. Dry bites are not common: in the series of 152 bites (*H. hypnale* 122, *H. zara* 22 and *H. nepa* 8) reported by Rathnayaka (2017) there were only nine (5.92%). A haemorrhagic blister at the bite site and painful, tender regional lymphadenopathy are the commonly encountered local effects, which, when present may be useful clinical features to identify the biting snake as hump-nosed viper when the biting snake is not available for identification. However, it is a nonspecific feature and is present following many other vipersid bites as well.

Systemic effects are rare and their occurrence is sporadic and unpredictable. In a series of 1,543 patients with hump-nosed viper bites observed in five hospitals, only 67 (4.34%) patients developed systemic effects with 2 (0.1%) deaths (Wijewantha & Sellahewa, 2010). The systemic effects varied from coagulopathy and nephropathy to neurological manifestations. Fifty-nine (3.8%) patients had only coagulopathy and they received either intravenous isotonic saline to ensure adequate urine output i.e. 0.5 ml/kg /hour or 15 ml/kg of fresh frozen plasma (FFP). None of the patients who had coagulopathy developed renal failure in this series.

Haemostatic dysfunction and acute kidney injury (AKI) are the commonest and most important systemic effects associated with mortality. Overt bleeding manifestations like haematuria, hematemesis and bleeding per rectum are rare but coagulopathy is often detected by the 20WBCT. The precise nature of the coagulopathy is poorly understood, it is most likely due to venom induced consumption coagulopathy.

**Acute Kidney Injury**

The primary cause for mortality following hump-nosed viper bite is from complications associated with coagulopathy and acute kidney injury (AKI) (Sellahewa, 2013). All who develop AKI have coagulopathy which can be detected before clinical and biochemical features of AKI become apparent. It is possible that early correction of coagulopathy with FFP could prevent AKI and related adverse clinical outcomes.

The association of coagulopathy with AKI after hump-nosed viper bite is well recognised (Varugunam, 1970; Dharmaratne et al, 1988-89; de Silva et al, 1994; Premawardena et al, 1996 & 1998; Maduwage et al, 2011a; Herath et al, 2012;). It is likely that common pathophysiological mechanisms are responsible for both these important systemic complications of hump-nosed viper bite. AKI is caused primarily by nephrotoxicity rather than myoglobinuria or haemoglobinuria. The procoagulant effects and venom induced consumption coagulopathy can also contribute to renal injury by the deposition of fibrin in the renal microcirculation and microvascular... coagulopathy is the earliest systemic manifestation among all patients who develop AKI.
Bites commonly cause only local swelling. Occasionally a haemorrhagic blister at the bite site. Less frequently tender regional lymphadenopathy. Rarely systemic effects such as coagulopathy, thrombotic microangiopathy (TMA), acute kidney injury and mild neurotoxicity.

Venom induced nephrotoxicity ranges from acute tubular necrosis, focal segmental glomerulosclerosis, and cortical necrosis to interstitial nephritis with the clinical manifestations of both acute and chronic renal failure. The mechanisms of renal injury have not been clearly elucidated. Both direct nephrotoxicity (Gunatilake et al, 2003) and adverse renal consequences of coagulopathy such as venom induced consumption coagulopathy and thrombotic microangiopathy (Herath et al, 2012; Karunatilake et al, 2012; Rathnayaka et al, 2017) are implicated. Interestingly coagulopathy was the earliest systemic manifestation among all patients who developed AKI. Arguably early correction of coagulopathy may prevent AKI.

Coma, external ophthalmoplegia, severe diarrhoea, and shock are some of the less commonly identified systemic effects of envenoming.

**Management of a patient bitten by Hypnale spp.**

As remarked earlier, dry bites are rare (Rathnayaka, 2017). Patients with a history of being bitten by a hump-nosed pit viper with puncture marks should be admitted for observation. Most will develop pain at the bite site and possibly a haemorrhagic blister. Observation for at least 48 hours is advocated owing to the possibility of delayed manifestations of coagulopathy that can have serious consequences.

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Antibiotics are not indicated unless there is local sepsis. Hump-nosed pit vipers commonly inflict their bites on the extremities with the risk of severe local tissue destruction. Bites on the digits of the hand and foot if so affected may result in tapering of the fingers and toes, or even require amputation (Ariaratnam et al, 2008). Puncturing of the blisters early with prophylactic antibiotic administration and elevation of the limb have been found to improve recovery.

The 20WBCT detects coagulopathy and can be used as an early predictor of systemic envenoming. Owing to the rarity and unpredictability of systemic manifestations and the recognized potential for a fatal outcome in patients with coagulopathy it is prudent that 20WBCT is monitored in all envenomed patients irrespective of the clinical status at presentation. Patients who develop coagulopathy as evidenced by a positive 20WBCT should be selected for intensive monitoring and aggressive therapy aimed at the early detection and treatment of venom induced consumption coagulopathy and thereby retard the onset of AKI.

The currently available snakebite antivenom is not effective in hump-nosed viper envenoming and should not be used (Maduwage et al, 2011b). Expectant treatment is the only option to offer patients. Those who develop coagulopathy may progress to AKI and would require haemodialysis.
Observe all patients for 48 hrs after hump-nosed viper bites even if the initial presentation is innocuous
Perform 20WBCT 6 hrly for 48 hrs
Select those with positive 20WBCT for specific therapy with FFP
   - The objective of FFP is the prevention of AKI as early correction of coagulopathy can prevent AKI
Hydrate adequately with isotonic saline and ensure adequate urine output of 0.5ml/kg body weight/hour
Currently available antivenom is ineffective for hump-nosed viper and should not be used

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Bibliography


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