RUSSELL’S VIPER BITES and their MANAGEMENT

_Daboia russelii_ (Shaw & Nodder, 1797)
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The Russell’s viper, _Daboia russelii_, is one of the most medically important snakes in the region (Kasturiratne et al, 2008, Alirol et al, 2010). Its bites cause death in 2% to 5% of instances, accounting for the majority of fatal snakebites in Sri Lanka (Kularatne, 2003).

It is a true viper distributed throughout the island except at extreme elevations over 1500 m above sea level. It is common in all types of habitats including rain forests, scrub jungles, grass lands and farm lands, and is particularly abundant in dry zone forests and paddy cultivations. The snake grows to 1.3 m in length and has a sub-triangular shaped head. The dorsum of the head is covered with numerous small scales and has a pointed white coloured “V-shaped” mark on the head, the body being light brown with a series of three well defined, dark brown, oval blotches arranged longitudinally.

The account that follows is based on published literature, the articles being listed in the bibliography.

Demography and epidemiology

Most Russell’s viper bites (63%) occur in the day time, between 6 am and 6 pm (Silva et al, 2016b). However, it is essentially a nocturnal snake and numerous bites occur at dusk and in the early hours of the night, usually on foot paths, roads and home gardens (Kularatne, 2003). Daytime bites are reported in paddy fields, where Russell’s vipers are abundant, during the harvesting seasons. Bites are reported throughout the year due to its distribution over a wide range of habitat types. Most of the Russell’s viper bite victims (70-80%) are males and the majority of the patients (77%) are between the ages of 10-40 years (Kularatne, 2003, Silva et al, 2016b). Forty-one percent of the bites occur in paddy fields, followed by foot paths and home gardens. Most of the bites are at the level of the ankle or below (86%) followed by the leg (Silva et al, 2016b).

Clinical manifestations

A wide range of clinical effects have been reported in Russell’s viper envenoming. Dry bites are not common: all patients developed at least local effects (Kularatne, 2003, Silva et al, 2016b). Severe pain and continuous oozing of blood from the fang marks is reported. Local swelling was present in 92% of the patients. Thirty five percent of the patients developed lymphadenopathy. Nausea, vomiting and abdominal pain seen in 91% of victims were non-specific systemic effects (Silva et al, 2016b).

Coagulopathy is the commonest (81%) systemic effect in Russell’s viper envenoming with prolonged clotting time (PT/ INR, aPTT) or depleted clotting factor levels (fibrinogen, factor V and X) and systemic bleeding (Maduwage et al, 2014, Isbister et al, 2015). The commonly seen systemic bleeding
manifestations are haematuria (50%), gum bleeding (22%) and haematemesis (10%) (Silva et al, 2016b). Acute kidney injury is a potentially fatal systemic effect of Russell’s viper envenoming seen in 4 – 18% of patients. Whereas most will recover with dialysis, a minority will progress to chronic kidney disease.

Intra-cranial bleeding and infarcts are not uncommon, occurring in any part of the brain, infarcts being commoner than haemorrhages (Gawarammana et al, 2009; Kularatne, 2003).

Neurotoxicity is a common systemic effect and 53% of patients developed clinically detectable signs (Silva et al., 2016b). Ptosis (partial or complete) and blurred vision were the earliest neurological features in most patients, followed by ophthalmoplegia. No facial, jaw, neck, bulbar, respiratory, or limb muscle weakness was reported following Russell’s viper envenoming (Silva et al, 2016b). Neurotoxicity developed within 8 hours of the bite and in 80% of individuals resolved within 3 days (Silva et al, 2016b).

Muscle tenderness and myalgia is commonly (90%) reported in Russell’s viper envenoming (Silva et al, 2016a). Hyperkalaemia is reported in 6.5% of patients, as a result of damaged muscle fibres leaking potassium. Hyperkalaemia may develop insidiously and vigilant monitoring is necessary to detect it early as otherwise, sudden cardiac arrest may occur. An ECG is the most specific diagnostic tool for hyperkalaemia which should agree with the serum potassium level (Kularatne, 2013). Cardiotoxic effects (3-12%) also have been reported (Kularatne, 2003). Combinations of more than one systemic effect are commonly seen in envenoming by this snake.

A small percentage of Russell’s viper envenomed patients develop chronic kidney injury a few weeks after the bite and continued clinical follow-up is important to detect such a condition early (Herath et al, 2012). Abdominal pain is an early non-specific symptom in Russell’s viper envenoming associated with later development of systemic effects such as coagulopathy, neurotoxicity and renal injury. Therefore, abdominal pain in Russell’s viper bites could be used as a predictor of future development of systemic envenoming (Kularatne et al, 2014).

Management of a victim of Russell’s viper envenoming

A patient with proven Russell’s viper envenoming should be managed as a medical or paediatric emergency. The general information for managing snakebite given in the SLMA Snakebite Management Guidelines, 2017, should be followed. Some recommendations are given below.

Investigations in proven or suspected Russell’s viper bite

Whenever the facilities are available coagulopathy should be detected by estimating the prothrombin time (PT)/International Normalized Ratio (INR), activated partial thromboplastin time (aPTT) or fibrinogen in blood samples.

When the facilities are not available for these tests, assess coagulopathy by the 20-minute Whole Blood Clotting Test (20WBCT). Follow the instructions in the SLMA snakebite management guidelines (section Initial Assessment) for performing this test as strict adherence to the standardised procedure is critical for accurate test results.
Blood urea, serum creatinine and serum electrolytes should be estimated to assess and monitor renal functions.

When cardiotoxicity is suspected, perform an ECG.

US scans of the abdomen have a place in detecting acute or chronic kidney injury. US scan of abdomen and CT of brain play a role in the detection of intra-abdominal and intra-cranial bleeding respectively.

Chest X-ray is indicated for acute respiratory distress syndrome.

Other investigations will depend on the type of organ and system involvement following Russell’s viper envenoming.

Antivenom treatment

Indian polyvalent antivenom is indicated for Russell’s viper envenoming, preceded by a prophylactic dose of adrenaline (see Guidelines, 2017) to reduce the rate of acute reactions. An initial dose of 20 vials is recommended. Each vial of antivenom is reconstituted with 10 ml of injectable water provided by the manufacturer. The total 200 ml of antivenom (20 vials) should be made up to 500 ml with 300 ml of normal saline and administered intravenously over one hour. Repeat tests of coagulability in 6 hours and if the blood is still incoagulable consider repeating antivenom in a dose of 10 vials.

*Recent research (Isbister et al, 2015; Maduwage et al, 2014) demonstrates that 20 vials of antivenom is adequate to neutralize the venom delivered by a Russell’s viper effectively. Recovery of clotting time and clotting factor levels takes more than 24 hours after the initial dose of antivenom. The authors, therefore, are of the view that repeated doses of antivenom at frequent intervals are not indicated in Russell’s viper envenoming. However, the considered opinion of the authors of the Guidelines, 2017 is that coagulability should be assessed and further antivenom considered every six hours.

Other treatment

Peritoneal or haemodialysis may be required for acute renal injury. Necessary supportive management is indicated depending on other systems involved.

Would fresh frozen plasma help? A study in 2013 showed that FFP administered after antivenom hastened the recovery of coagulopathy in Australian snakebites (Isbister et al, 2013). However, a similar study in Sri Lanka where FFP was administered after antivenom showed no beneficial effect in hastening the recovery of the coagulation defect in Russell’s viper envenoming (Isbister et al, 2017). This question is still open for debate and more studies on the subject are awaited.
Bibliography


