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Direct nephrotoxic effects produced by venoms of Sri Lankan cobra, Russell's viper and hump nosed viper

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Abstract

Nephrotoxicity is the principal cause of death following Russell's viper envenomation. Envenomation following the bite of several other snakes is also known to cause nephrotoxicity. The nephrotoxicity can be due to direct effects of venom or secondary to circulatory disturbances (eg. ischaemia), which these patients often manifest. As separating out the contributions of direct toxic effects and ischaemic effects are difficult in the *in vivo* situation, experiments were carried out using the kidney slice model to study and compare the direct toxic effects of venom of cobra, Russell's viper and hump nosed viper.

The effect of cobra venom (CV) on rat kidney slices and the effects of Russell's viper venom (RVV) and hump nosed viper venom (HNVV) on rabbit kidney slices were examined. Healthy male animals were anaesthetized and kidneys were harvested. Kidneys were decapsulated, bisected and sliced. Rat kidney slices were incubated with CV and rabbit kidney slices were incubated with RVV and HNVV for different time periods. Rat and rabbit kidney slices were incubated with 0.9% sodium chloride as the control. At the end of each observation period kidney slices were preserved for light and electron microscopy (LM and EM).

When CV was used, complete necrosis was seen in proximal and distal convoluted tubular cells (PCT and DCT). When rabbit kidney slices were incubated with RVV for 4 hours there was complete necrosis of glomeruli and PCT with the preservation of the basement membrane. LM and EM changes were mostly confined to PCT when HNVV was used.

The results of this experiment provide evidence that the venoms studied produce direct damage on renal tissue. Different areas of the nephron are differentially susceptible to the effects of the three venoms.

Key words : direct nephrotoxicity, Russell's viper venom, cobra venom, hump nosed viper venom, kidney slice model

Introduction

The death rate due to snake bite envenomation in Sri Lanka is one of the highest in the world (1). These fatalities are often caused by cobra, Russell's viper and the common krait. Though cobra and krait venom are more potent than Russell's viper venom, Russell's viper is the most dreaded snake in the island (2) and it is responsible for 40% of deaths due to envenomation (1).

Although death following Russell's viper envenomation could be due to different reasons (shock, haemorrhage, disseminated intravascular coagulation, neurotoxicity, respiratory failure or acute renal failure), nephrotoxicity leading to acute renal failure has been identified as the principal cause (1). In one study it was found that envenomation by Russell's viper contributes for 49% of cases of acute renal failure reported at the National Hospital of Sri Lanka (3). Envenomation following the bite of several other snakes (e.g. cobra, pit vipers and sea snake) is also known to cause nephrotoxicity (4, 5).

The development of nephrotoxicity in envenomation can be due to direct effects of venom or secondary to circulatory disturbances (ischaemia), which these patients often show. Separating out the contribution of direct

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