

Table 74.2 Progressive onset of major systemic symptoms and signs of untreated envenomation (in massive envenomation or in a child, a critical illness may develop in minutes rather than hours)

< 1 h after bite

Headache
Nausea, vomiting, abdominal pain
Transient hypotension associated with confusion or loss of consciousness
Coagulopathy (laboratory testing)
Regional lymphadenitis

1–3 h after bite

Paresis/paralysis of cranial nerves, e.g. ptosis, double vision, external ophthalmoplegia, dysphonia, dysphagia, myopathic facies
Haemorrhage from mucosal surfaces and needle punctures
Tachycardia, hypotension
Tachypnoea, shallow tidal volume

> 3 h after bite

Paresis/paralysis of truncal and limb muscles
Paresis/paralysis of respiratory muscles (respiratory failure)
Peripheral circulatory failure (shock), hypoxaemia, cyanosis
Rhabdomyolysis
Dark urine (due to myoglobinuria or haemoglobin)
Renal failure

natural
history

envenomation

effects

Neurotoxins

Presynaptic and postsynaptic neuromuscular blockers present in all dangerous venomous snakes. May cause paralysis. Postsynaptic blockers readily reversed by antivenom. Presynaptic blockers are more difficult to reverse, particularly if treatment is delayed. Some presynaptic blockers are also rhabdomyolysins.

Prothrombin activators

Present in many important species. Cause disseminated intravascular coagulation with consumption of clotting factors including fibrinogen. Intrinsic fibrin(ogen)lysis generates fibrin(ogen) degradation products. Significant risk of haemorrhage.

Anticoagulants

Present in a relatively small number of dangerous species. Prevent blood clotting without consumption of clotting factors.

Rhabdomyolysins

Some presynaptic neurotoxins also cause lysis of skeletal and cardiac muscle. Apart from loss muscle of mass, may cause myoglobinuria and renal failure.

Haemolysins

Present in a few species. Rarely a serious clinical effect.