Blindness and autonomic instability following Russell's viper bite

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Abstract

Autonomic instability is a rare complication following elapid bites. Blindness too is a rare complication following Russell's viper bite and is most likely due to cerebral infarction or direct ocular toxicity. We report a case of a young male from Sri Lanka who developed both transient blindness and autonomic instability following severe envenomation by a Russell's viper bite.

Introduction

The Russell's viper (*Daboia russelii*) is well-known for its deadly bite and toxic venom. It is distributed over many south Asian countries including Sri Lanka and is a leading cause of fatal snake bite. It is responsible for around 30-40% of all snake bites and causes the most number of life-threatening bites of any snake found in Sri Lanka [1].

Bites by Russell's vipers commonly occur in and around paddy (rice) fields and close to footpaths mostly at dawn and dusk, affecting a large number of agricultural workers therefore it is considered an important occupational hazard in Sri Lanka [1].

Clinical features of Russell's viper envenomation are local envenomation 92%, coagulopathy 77%, neurotoxicity 78% and renal failure 18%, either in various combinations or as isolated manifestations [1]. Other complications are thrombotic microangiopathy, rhabdomyolysis, myocarditis, myocardial necrosis and rarely ocular complications [2].

Causes of death include shock, intracranial haemorrhage, massive gastrointestinal haemorrhage and acute tubular necrosis or bilateral renal cortical necrosis.

Autonomic dysfunction has been reported in snake bite, mostly following bites of elapids. This occurs as a complication of neurotoxicity [3,4]. Dysautonomia following viper bites has rarely been reported.

Blindness following viper bite has been reported in several patients. There are reports of patients developing bilateral blindness secondary to a bilateral posterior circulation ischemic stroke following Russell's viper envenomation [5].

Very rarely bilateral retinal haemorrhage has been reported, leading to blindness following Russell viper envenomation [2].

We report a case of severe envenomation following Russell viper bite, causing dysautonomia and blindness secondary to macular oedema and intraocular haemorrhages.

Case report

A 48 year old previously healthy male presented to Colombo North Teaching Hospital, one-hour following Russell's viper bite to the hand with fang marks and bleeding from the bite site (snake identified at emergency treatment unit, from an image of the dead snake). On admission he complained of generalized abdominal pain and was found to be hypotensive with a blood pressure of 70/50mmHg without any evidence of neurotoxicity or any bleeding manifestations. He was resuscitated and given 20 units of polyvalent anti-venom (AVS) and continually monitored. He was started on IV noradrenaline infusion at a rate of 0.2µg/kg/min, as he was having persistent hypotension. 30 minutes later he started to develop bilateral ptosis with neck muscle power of 4/5 and single breath count of 7.

After 1 hour he developed gross haematuria, therefore 10 units of AVS was repeated. His single breath count dropped to 4 and neck muscle power was 3/5 which necessitated elective intubation and transfer to the ICU where he was ventilated under deep sedation. Whole blood clotting test (WBCT) was prolonged even after 6 hours and another 10 units AVS was given. His blood

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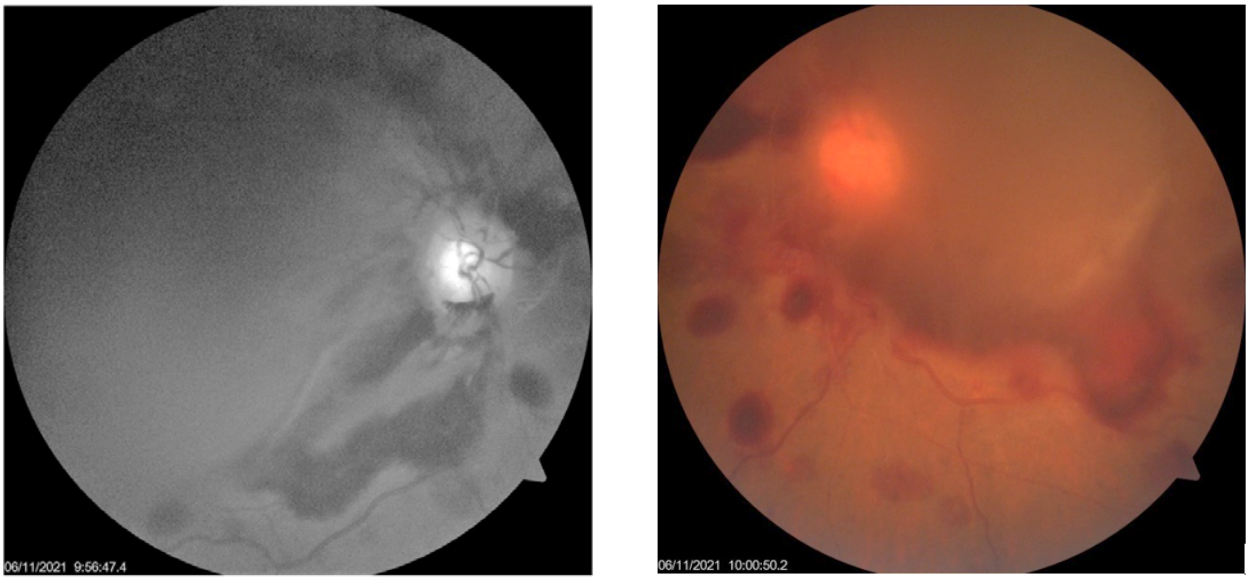

Fundoscopy image from our patient – with permission.

pressure gradually picked up and we were able to tail off his noradrenaline infusion over the course of 24 hours.

By the next day his average blood pressure recording was 140/100 mmHg with a pulse rate of around 80 beats per minute. While he was on deep sedation with midazolam infusion the multi-monitor in the ICU recorded fluctuating blood pressures, serial reading of 140/70mmHg, 115/60 mmHg, 170/90mmHg, 100/60mmHg, 220/130mmHg, 150/80 mmHg, 210/130mmHg, 166/107mmHg. This was associated with a tachycardia of 115-130 beats per minute.

This trend of gradual rising and sudden drop of blood pressure with tachycardia persisted through 72 hours even while he was well sedated, after this period blood pressure readings plateaued around 140/80mmHg.

During the ICU stay he also developed oliguric acute kidney injury which needed dialysis, 7 cycles were given in all. Blood picture showed evidence of microangiopathic haemolytic anaemia with a haemoglobin of 7.5g/dl and platelet count 21000u/L, which was managed with fresh frozen plasma and blood transfusions during heamodialysis. He had a creatinine phosphokinase level of 8000 and elevated cardiac troponins with a normal 2DECHO cardiogram.

He was extubated after 5 days of ventilator support. When he was able to talk he complained of blindness which had been there since just after admission to hospital, fundal examination revealed retinal haemorrhages in both eyes complicated with macular oedema. Optic coherence tomography was performed and images of the retina showed vitreous haemorrhages and macular oedema for which he was prescribed ketorolac tromethamine eyedrops.

He was discharged after a 20 day hospital stay, In summary he had severe envenomation following Russel's viper bite, complicated with coagulopathy, microangiopathic haemolytic anaemia, neuropathy with autonomic instability, nephropathy, myotoxocity with rhabdomyolysis, cardiomyotoxicity and transient blindness due to retinal and macular oedema with intraocular haemorrhages.

At the last follow up, 6 months after the initial presentation he remains asymptomatic. His vision had improved gradually and visual acuity was 6/6 during the last review.

Discussion

We report a case of severe envenomation following Russell viper bite, leading to dysautonomia and transient bilateral blindness due to retinal and macular oedema with intraocular haemorrhages.

Dysautonomia has previously been reported in cases with envenomation following bites of elapids like krait and cobra, these effects on the autonomic system manifest as transient tachycardia with fluctuating blood pressure which sometimes warrants use of intravenous antihypertensive treatment [3, 4]. Autonomic dysfunction is mostly self-limiting and needs no specific management, apart from ASV and supportive care.

Even though reports of dysautonomia following Russell viper bite has rarely been reported, we presumed that the autonomic dysfunction seen in our patient was due to the neurotoxic effects of Russell viper bite.

Bilateral blindness secondary to a bilateral posterior circulation ischemic stroke has been reported as a neurologic manifestation following snake bite [5].

Snake venom contains proteins, enzymes, and chemicals with cytotoxic effects, neurotoxins, coagulants and anticoagulants. Manifestations of each snake bite depend on the specific toxins that constitute its venom. Viper venom contains anti-haemostatic factors which can lead to acute fibrinolysis, severe thrombocytopenia and damage to the vascular endothelium causing breakdown of permeability barriers resulting in fluid leakage and haemorrhage.

Proteolytic enzymes like hyaluronidase and collagenase, found in viper venom, may cause disruption of retinal veins resulting in retinal haemorrhages.

Ocular complications following Russell viper bite have been reported in cases from India. Of those described, blepharoptosis, muscle palsies, ocular haemorrhage (conjunctival, anterior chamber, vitreous or retinal) lid oedema, conjunctival chemosis, retinal and optic nerve oedema, pupillary changes, optic neuritis, optic atrophy, acute angle closure glaucoma, central retinal artery occlusion (CRAO), macular infarction, exudative retinal detachment, globe necrosis, endophthalmitis, and blindness caused by cortical infarction have been reported.

Bilateral retinal haemorrhage has been reported as an infrequent ocular effect caused by snakebite [2].

In our patient fundoscopy showed macular oedema with haemorrhage and bilateral vitreous and pre-retinal haemorrhages.

These effects could be a result of a combination of multiple factors, namely increased vascular permeability, bleeding due to venom induced consumptive coagulopathy, as well as transient severe hypertension caused by autonomic instability.

Author contributions

KRF was involved in gathering the patient's details, investigations, literature search, drafting the manuscript and correspondence. SJ was involved in patient management, critical decision making and literature review. AP was involved in literature review, formatting, revising and in the final draft of this article.

Conflicts of interest

The authors declare that there are no competing interests.

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