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CASE REPORT

FIRST TWO CASES OF RUSSELL'S VIPER (*DABOIA RUSSELII)* BITE, A RE-EMERGED CAUSE OF ENVENOMATION IN BANGLADESH

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Abstract:

The first two cases of Russell's viper bite (RV) presented in contemporary period from Chapainwabgonj and Patuakhali districts of Bangladesh in 2013. The unfortunate death of both the cases indicates the severity it can produce and difficulties in its management in resource limited settings. Treatment seeking from traditional healer, sub-district hospital could not provide first dose of AV before referral and delayed arrival to hospital are concerned to be addressed to prevent death from bite by RV in future. Basic preventive measures such as careful while working and walking in the field and unnecessary handling of snake could avoid bite in both the cases.

Keywords: *Russell's Viper, Daboia russelii , Envenomation*

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Introduction:

Snakebite a common cause of morbidity and mortality in rural Bangladesh mostly among the poor farming community. Among the venomous bite cobra, krait andviperidae snakes are common, the green pit viper being the commonest in certain hospital for example, Chittagong Medical College Hospital.[1,2,3]A nation-wide community-based survey conducted 14 years before in Bangladesh recorded approximately 700,000 snake bites/year with about 6,000 fatalities.[4]Russell's viper (Daboia russelii) is found in Asia- throughout the Indian subcontinent, much of South East Asia, Southern China and Taiwan.[5]The species was named in honor of Patrick Russell (1726–1805), a Scottish herpetologist; and the name of the genus is from the Hindi word meaning "that lies hid", or "the lurker".[6]The snake become very aggressive in nature when disturbed or threatened and sodeadly that Sri Lankans consider this snake as personification of devil.[7]Althoughthe Russell's viper (locally called 'Chandrabora' or in some part 'Uloobora') is described to be present in Rajshahi and Khulna division for long[8,9]there has been no scientific evidence of bite happened in Bangladesh in distant past. Given this background this is probably the first two cases of Russell's viper bite observed by clinicians of two

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different health facilities in Bangladeshafter more than several decades. These casesrepresent there-emerging cause of envenomation in Bangladesh.

Case Report:

Case 1:

A 20-year-old male from Nachole sub-district, Chapainawabganj district on the western part of Bangladesh (Figure 1), was bitten in his right lower forearm by a snake at 11.00 hours 2 June,2013 while he was trying to capture it as a bet with his friends that he can capture live snake. Earlier he watched some documentaries on catching live snakes on TV and felt quite confident about catching it. When he lifted the snake after grabbing the tail, it made ca hissing sound and bit him. He and his friend thought that it would be a non-venomous python as it has a heavy body and chain like markings so did not seek any medical advice immediately after the incident.But after an hour when his forearm became swollen, he went to a local 'ozha' (traditional snake bite healer), who assured him that no envenomation had occurred as it was a 'python' but put two tight tourniquets in his right forearm and mid arm.Ten hours later, when blood started to ooze from the bite site and swelling increased, he rushed to Rajshahi Medical College Hospital (RMCH), Rajshahi. They brought the digital image of the snake taken through mobile device (Figure- 2) before they had beaten the snake and threw it away.

Figure 1: *Map of Bangladesh showing distant location of Case- 1, Nachole Upazila, Chapainawabganj district & Case- 2, Kalapara Upazila, Patuakhali district found in 2013.*

Figure 2: *Offending 'Chandrabora', Russell's viper (Daboia russelii) involved in the case- 1*

On arrival at RMCH on 3 June, 2013 at 9.00 hours (22 hours after the bite) his right forearm was hugely swollen. There was no ptosis, external ophthalmoplegia or 'broken neck' sign. Even after removal of the tourniquet, his right radial and brachial pulses were not palpable, BP was 80/50 mm Hg, pulse was feeble, 110/min. There was constant oozing of blood from the bite site, gum bleeding, conjunctival chemosis andecchymosisdeveloped in the anterior chest wall. The twenty-minute whole blood clotting test (20 WBCT)was positive. Ten vials of polyvalent antivenom (27 hours post-bite), and IV fluid was started, and he was transferred to ICU in the same hospital. After a total additional 40 vials of antivenom over 08 hours, his bleeding stopped, and 20 WBCTwas negative.Lab tests: hib 8.9 mg/dl, TC WBIC 12,000 per cu mm with normal differentials, platelet count 40,000 cu mm, prothrombin time 22 sec (lab reference 12 sec) with INR 2.1; APTT 52 sec (lab reference 28-38 sec), D dimer 467 ng/ml (lab reference 250 ng/ml). Considering it as a case of disseminated intravascual coagulation one unit of platelet rich plasma was transfused (fresh frozen plasma was not available). Since the patient clinically developed compartment syndrome (intra-compartmental pressure could not be measured) of the right upper limb,fasciotomy was done. According to surgical note, patient did not lose significant amount of blood during the procedure. Despite fluid support, he became oliguric 360 ml over initial 24 hours stay at ICU), and urine was noted to be reddish in color. Serum CPK was 9800 IU/L, serum creatinine 7.1 mg/dL, serum k 5.6 mmol/L. Antivenom was continued up to a total of 50 vials as acute renal failure was not controlled and subsequently haemodialysis was started. His blood pressure (BP) and oxygen saturation continued to fall, and he became drowsy. He was intubated and despite all supportive measures, the patientdied(3 days post-bite) (Table - 1).

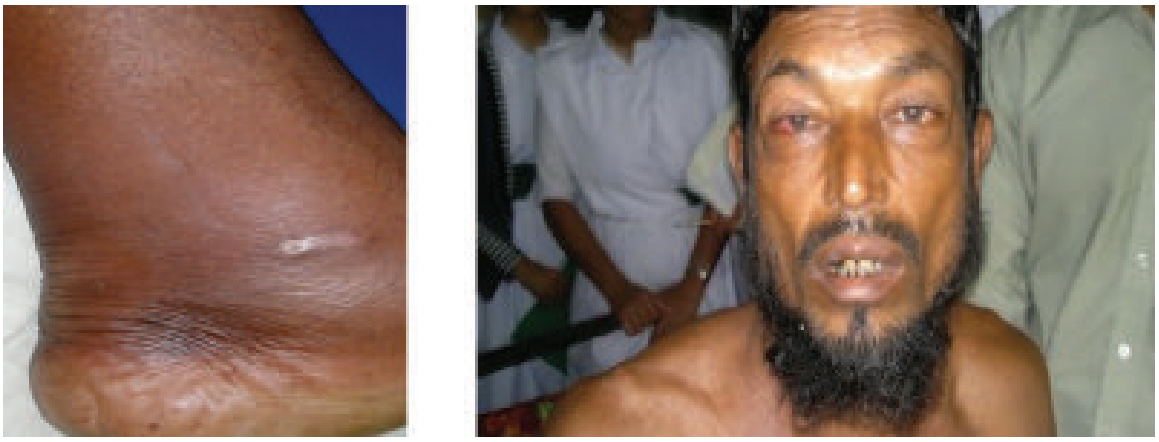
Note: The snake was identified initially on admission by the teambut discussed after around six months of the incident by one of the author(MRA) during a training session on management of snakebite at RMCH, Rajshahi.

Case 2:

A 46-year- old male from Kuakata, Kalapara sub-district, Patuakhali district in the southern coastal part of Bangladesh (Figure 1)referred from Patuakhali district hospital toDhaka Medical College Hospital(DMCH), Dhakaon 30thNovember, by one of the author(FMAR).The mancomplained of snakebite on 27th November followed by development of multiple ecchymoses on various parts of the body, scanty and high coloured urine for 2 days and passage of black tarry stool for single episode. According to the statement of the patient's attendant, while walking through paddy field he was bitten suddenly by a snakeon his left leg below the ankle. He described the snake as having a triangular head and spiral striped body. There was bleeding and pain from the bite site. He had put a tourniquet in his left thigh and rushed to local sub-district hospital immediately. There hereceived Inj. tetanus toxoid (TT) and (tetanus Immunoglobulin (TIG) and as there was no systemic effect he was discharged after a couple of hours' observation. He stayed the night at home but became restlessness and felt little drowsy and his eyes were heavy.

The neighbor took him toPatuakhalidistrict hospitaland diagnosed as a neurotoxic snake bite as evidence by ptosis and ophthalmoplegia.However, conjunctival chemosis, ecchymosis and history of bleeding from bite site was not consistent with locally prevalent neurotoxic snakes- cobra or krait. Local examination revealed a bite mark below the ankle with swelling and having ecchymosis at swollen area.

The patient received 1st dose (10 vials) polyvalent antivenom at Patuakhali district hospital. His 20WBCT was found to be positive. Total count of WBC was 54.600/cummand platelet count 19200 /cumm, and serum creatinine 2.5mg/dl.Duringthe follow up external ophthalmoplegia disappearedbut ptosis was present.As the condition was deteriorating with unstable vitals(low BP and tachycardia) he was referred to DMCHon the morning of 30thNovember, 2013.



(a) (b)

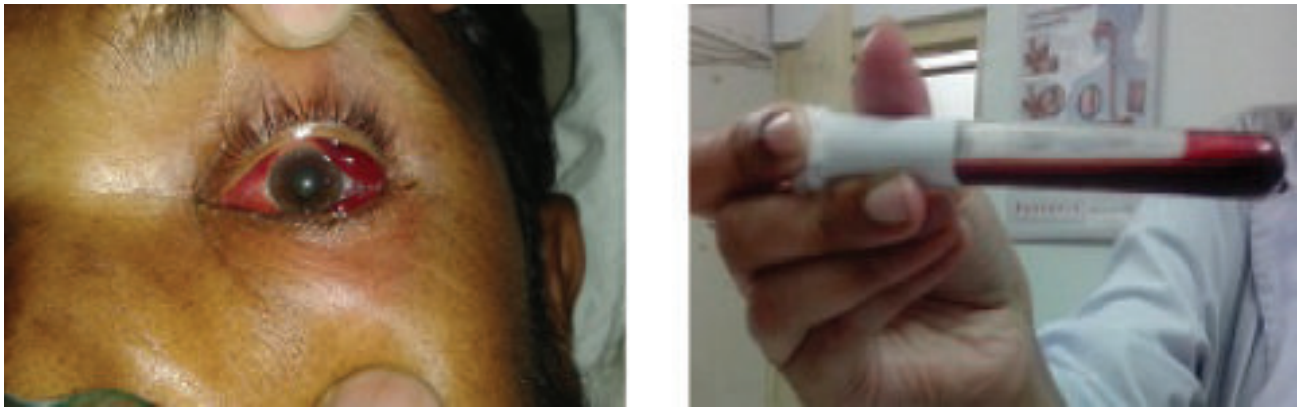
Figure 3: *(a) Fang mark with swelling and ecchymosisj, (b) Partial ptosis with conjunctival chemosis*

On admission at DMCH the patient had bilateral ptosis without anyother neurologicalmanifestationlike external ophthalmoplegia, difficulty in swallowing or complaints of double vision.However, he complained of gradual dimness of vision and another episode of melaena. On examination mild pitting edema, periorbital swelling, overt conjunctival chemosis and subconjunctival haemorrhage was noted. There was huge ecchymosis at his right shoulder and left forearm. Fundoscopy revealed bilateral retinal haemorrhage. He was afebrile, blood pressure was 80/50 mm of Hg, pulse-114 b/min and respiratory rate was 18 breaths/min. There was a small amount of urine after catheterization and it was dark in colour. 20WBCT was positiveeven on the 3rd day of snakebite.

The patient was shifted to ICU as the BP was decreasing persistently. Hb was 4.8 gm/dl, ESR 120 mm in 1st hour, RBC 1.8 million per cubic mm, TC of WBC 15000/cu mm and platelet count was less than 10000/cu mm.Prothrombin time was 60 secs with INR 5.59,APTT was 68 sec and FDP was 20 microgram/ml, blood urea 230mg/dl, serum creatinine 6.43 mg/dl, S. Na 124 mmol/l, K 6.4mmol/L Cl 86 mmol/l and HCo3 24 mmol/l. The diagnosis was madeas DIC and multi organ failure with shock possibly due to Russell's viper bite (Table- 1).

Inj. polyvalent antivenom 10 vialswas given in the ICU along with necessary supportive care (Inj. normal saline, inj.Nutridex + soluble insulinto combat hyperkalemia, inj. Sodibi-carbonate, inj. Frusemide, inj. Dopamine, inj. Noradrenaline) and parenteral antibiotics.

Nephrological consultation was sought and despite all efforts his condition was rapidlydeteriorated. His vitals become unstable within 4hours of shiftingin ICU. Respiratory support with mechanical ventilation was started but there was no recordable BP. The patient died inICU after few hours. The dialysis could not be started during this preparatory management. Autopsy was not done on this patient due to court exemption.



(a) (b)

Figure 4: *(a) Subconjunctival haemorrhage and chemosis, (b) Positive 20WBCT*

Table-I

Two initial cases of Russell's viper bite with clinical syndrome,treatment and outcome in 2013

Case No	Date of attendance	Age/sex	Hospital arrival	Envenomation	Specific treatment	Outcome (Survival/Death)
1	03.06.13	20/M	6 hrs after bite	Local envenomation, Coagulopathy, ?Myotoxicity, Acute kidney injury (AKI)	Antivenom-50 vials, Fasciotomy	Death
2	30.10.13	46/M	Patuakhali-12hrs after bite DMCH-4thday	Local envenomation, Neurotoxicity, Coagulopathy, Myotoxicity, AKI Intracranial haemorrhage?	Antivenom-20 vials	Death

Discussion:

Russell's viper*locally known as* 'Chandrabora', 'Uloobora', 'Guiyyapora' are found in Rajshahi, Kushtia,Jashore and Khulna[8,].It was said to be available in the present territory of Bangladesh in early 20th Century.[9]RVthought to be near extinct in Bangladesh,we were searching for a victim of RV from Bangladesh since 1993 through inspiration of one of the authors (MAF). In India and Myanmar, Russell's viper is as high as 70% of the venomous snakebite, and commonest cause of death due to snakebite in Sri Lanka.[10,11] There has beenno report of bite by Russell's viper in Bangladesh before 2013. We tried togather information retrospectively while discussing about RV bite ina training session at Rajshahi Medical College and thus revealed the first case of Russell's viper bite in Bangladesh with unfortunate death (Case-1, Table- 1).The existence of Russell's viper was described in the literature in Bangladesh[8,12]but the clinical incidence with certainty was lacking as the hospitalshave not reported any such case in several decades although there is no good reporting system of snakebite in place. The first case was disclosed as an unnamed venomous bite from RajshahiMedical College during the training session (through the case discussion between the authors, MZH, MAHA, MRA and FRC) and the neighborhood of bite area repeatedly mentioned the local name of the snake as 'Uloobora' or 'Chondrabora.

In the first case the victim thought that the snakecould be a non-venomouspython and tried to catch it. The wild life expert also described similarity in appearance between pythonand Russell'sviper at the first sight[13] with the possibility of potential mistake by the community. The index case had severe coagulation failure and possible myotoxicity (had raised CPK several folds) with progressive AKI which did not respond to repeated antivenom doses. The second case was from Patuakhali where the local name of the snake was 'gyuiyyapora'. In Patuakhali hospital he was shown the picture of Russell's viper for identification which he agreed. The Russell's viper hastriangular flat head flat, covered with small scales,body stout, short and flattened dorso-ventrally. Scales strongly keeled, tail short. Colour is light-brown, head with two large black spots at base and a light V-shaped mark with its apex on top of snout.[13,14]The size varied from 1meter to 1.85meter(Fig-2).The 2ndcase presented with clinical syndrome of Russell's viper,althoughthe snake was not brought to hospital but the pictorials for identification were consistent with RV.

The local swelling, bleeding manifestationswith AKI and neurotoxicity are clinical features found in patients inSri Lanka and south India following Russell's viper bite.[11,14,15]Our second case had some features of neurotoxicity (Fig-3b) initially followed by coagulation failure leading to bleeding. Features of neurotoxicity in victims of RV bite in Sri Lanka and south Indiaare drowsiness, paraesthesia, abnormalities of taste and smell, "heavy" eyelids,ptosis, external ophthalmoplegia, paralysis of facial muscles, nasal voice, regurgitation through the nose, difficulty in swallowing secretions, respiratory and generalized flaccid paralysis. Some of these features were observed in the second case (from Patuakhali) which is geographically distant from locality of the first case. Whether the species of Russell's viper in Patuakhali is different from the species from the northern territory is yet to be ascertained. The venom variation among the snake of different geographical areas of Russell's viper of the Western variety are described.[16] Although the neurological manifestations with local swelling are classical of elapidae bite (cobra), but conjunctival chemosis or widespread swelling are findings of Russell's viper bite

may be had associated 'capillary leak syndrome'(Fig 4a).[17] The fang mark is usually distinct in Russell's viper bite with bleeding from bite site as well as surrounding ecchymosis (Fig-3a).This description is also common to Russell's viper bite from Sri Lanka and Myanmar.[11,17]

The unique feature in both cases wasrestlessness. Although literature showed paraesthesia and drowsiness is common as part of cardiovascular toxicity, the restlessness was not commonly written in other observation. The second case was restless during all through of his illness which is difficult to explain as pain was not a constant feature. But as he was taken care of by traditional healer with multiple interventions, he may remain restless for long. He became drowsy as time goes by and at district hospital, he was found restless and sleepy. Ptosis and external ophthalmoplegia were observed which let the physician to start antivenom for neurotoxicity but the consultant (AFAR) was concerned about conjunctival suffusion and restlessness. The bed side 20WBCT was positive and so also the other parameters of coagulation defects like prolonged prothrombin timewere very much suspicious of some unusual snakebite not found in that area before. There was a lack of response although ptosis and ophthalmoplegia disappeared but the bleeding continued and the urine became high coloured and oliguria ensued which indicated the classical Russell's viper venom induced consumption coagulopathy and acute renal failure.[15,17] His blood pressure reduced quicklywhich could be due to either bleeding (concealed and revealed) or byeffects of cardiotoxin or might be associated acute adrenal failure.[17]

At DMCH the patient was on the 3rd day of envenomation with a rapidly deteriorating course having visual disturbance, dizziness, fainting, collapse, shockwhich is common in late phase of Russell's viper bite. The investigations were consistent with DIC and consumption coagulopathy having PT, APTT and FDP raised which indicates kallikrein like proteinases which may interact with the contact phase by activating coagulation factor XII.A highly specific activator of clotting factor V(RVV-V) derived characteristically from Russell's vipervenom.[15,17]Activator of factor X is detected (RVV-X) in the venom of *Daboirusselli* has also been shown to activate clotting factor.[15]Viperidae venoms contain serine proteinases which also convert fibrinogen into fibrin.[15] The progressive renal impairment with persistent bleeding indicates the severity leading to poor outcome as the second dose of antivenom was not also successful in reversingthe 20WBCT(Fig-4b). We could notdo a CT scan to rule out haemorrhage within CNS or echocardiography to see evidence of cardiac dysfunction. Despite all supportive management the patient had severe metabolic acidosis and died within 3 hrsof ICU stay. Surgical intervention can lead to more complications in patients with coagulation failure.

We are drawing the attention ofhealth care workers about the re-emergence of Russell's viper cases in Bangladesh after an absence of more than several decades. The reasons of re-emergence behind this could be multi-factorial. Climate change effect (more floods), de-forestation, change in ecology and biodiversity (extinction of animals who prey snakes) of the region, availability of food, lack of competitor in the environment,changes in snake behaviors, population and others are enhancing Russell's viper-human interaction.[18,19] Changes in venom composition of Russell's viper and evolutionary increase ofpotency and lethality of the venom are other interesting areas of future research. The hospitals in the hot spot areas should be kept well equipped and alert to ensure prompt management of cases and timely referral to higher centers. Production and use of monovalent anti-venom can certainly help better management of cases and reduce fatality in future.

Ethics: Written consent was taken from Case-2 for taking and using photographs for academic purpose and publication.

Conflicts of interest: None declared

Consent:
Informed consent was obtained from the patient for the publication of this case report.

Declaration:
The authors declare no conflict of interest.

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