**RESEARCH PAPER**

# A Unusual Case Report Of Juvenile Russels Viper Bite Causing Priapism

Dr.Sharath Chandra Reddy Turpu [1], Dr.V Padma [2], Dr.Vinatha MC [3], Dr.S.V. Sathyapriya [4], Dr.Jagadesh [5], Dr.V.VeeraVignesh [6], Dr.Bhavana Reddy [7], Dr. Snega R Laxmi[8]

*[1]Department of General Medicine, Sree Balaji Medical College, Tamil Nadu, Email Id- turpusharath@gmail.com, ORCID ID: 0009-0001-6314-367X*
*[2]Department of General Medicine, Sree Balaji Medical College, Tamil Nadu, Email Id - padmaramesh86@yahoo.com, ORCID ID: 0000-0002-9938-6462*
*[3]Department of General Medicine, Sree Balaji Medical College, Tamil Nadu, Email Id: vinathamadhukar@gmail.com , ORCID ID: 0000-0003-4317-6842*
*[4]Department of General Medicine, Sree Balaji Medical College, Tamil Nadu, Email Id : svsatz1994@gmail.com, ORCID ID: 000-0002-3397-291X*
*[5]Department of General Medicine, Sree Balaji Medical College, Tamil Nadu, Email Id jagasri11@gmail.com , ORCID ID: 0009-0007-9483-7301*
*[6]Department of General Medicine, Sree Balaji Medical College, Tamil Nadu, Email Id drveeravignesh@gmail.com, ORCID ID: 0009-0000-3437-1457.*
*[7]Department of General Medicine, Sree Balaji Medical College, Tamil Nadu, Email Id m.bhavanareddy12@gmail.com, ORCID ID: 0009-0004-3910-9713*

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**ABSTRACT**
Following a bite from a juvenile Russell's viper (Daboia russelii), a painful erection known as priapism developed quickly. Three hours after the antivenom was given to a 16-year-old boy, the priapism went away. The most likely cause of snakebite-induced priapism is the venom toxins' production of nitric oxide (NO), which causes the endothelium in the corpus cavernosum to dilate. Other processes may also be at work.We are very hopeful that this singular case report may spark new lines of inquiry into the etiology and clinical manifestations of envenomation caused by bites from Russell's vipers. While it is premature to make any firm predictions, future studies may potentially uncover opportunities to create candidate compounds based on venom that may cure male and female sexual dysfunction..
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## INTRODUCTION

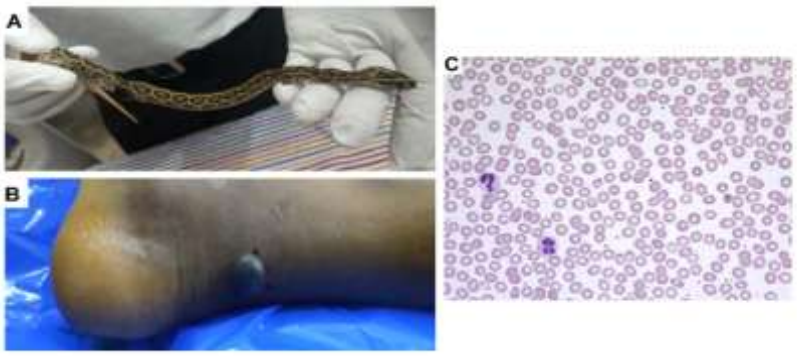
Worldwide, snakebite envenomation (SBE) results in approximately 140,000 fatalities and over 500,000 long-term impairments [1,2]. There are four main families that include more than 600 species of poisonous snakes [3]. The envenomation consequences of these many different species are varied and extensive, including myonecrosis, coagulopathy, flaccid paralysis, acute renal damage, PTSD, and many more [4,5]. In most cases, researchers believe that various venom components from various snake groups are responsible for these diseases. A teenage boy who was nibbled by a youthful Russell's snake (Daboia russelii) developed priapism, a rare occurrence. The majority of the literature on priapism following venomous chomps is restricted to the Brazilian meandering insect (Phoneutria nigriventer) and other spiders, despite examples involving other spiders and venomous invertebrates. Male mice were infected with the venom, which caused persistent priapism and frequently resulted in death [6].There does not seem to be any published information about priapism after bites from vertebrates, particularly snakes, as far as we are aware. As a result, we provide evidence of this unusual priapism following a Russell's snake nibble and propose conceivable causes.

### Presentation of a case

About 9:00 p.m. India Standard Time (IST), a male patient who was 16 years old was sent to the emergency room after suffering a snakebite on his right foot. The bite occurred at approximately 6:30 pm IST, when the patient was wandering in his yard. When the snake was killed and brought to their office, a herpetologist affirmed that it was a youthful Russell's snake (Fig. 1A). During the assessment, the casualty's important bodily functions were viewed as inside the ordinary range.His vital signs were steady, and he was alert, worried, afebrile, and cognizant. His systemic exam came out normal, but when we looked closer, we saw blisters, edema, and fang marks on his foot (Fig. 1B). We also felt painful lymph nodes in his right inguinal region. The genital exam showed an enlarged, swollen, and arched penis with a tight and sensitive corpora cavernosa that spared the corpus spongiosum and glans. The penis did not

**Author for Correspondence: drnaganand1971@gmail.com*

change color. It seemed like everything was OK with the testes and the prostate. The patient reported experiencing an unexpected and unanticipated penile erection within fifteen minutes of the snakebite, which he attributed to a spontaneous process. On top of that, he admitted to feeling some pain when he was getting this erection. He adamantly denied having ever had the desire for sex or had any prior experience with genital stimulation. Up until he got the antivenom, his erection persisted for another two hours. He has never abused drugs or been on any medications, and he has no co-morbidities. Neither he nor his family had ever tried any traditional remedies for perineal injuries. Autonomic symptoms such postural hypotension, piloerection, or profuse sweating were not present. His activated partial thromboplastin time and prothrombin time were both longer than normal. Table 1 shows that all of the other biochemical, metabolic, and hematopoietic markers were within normal range. It was found that the results of a urine test had nothing to do with this problem. No abnormal red blood cells were discovered during the microscopic examination (Fig. 1C). None of the screening tests showed any signs of malaria or sickle cell anemia. The casualty has been given 100 milliliters of polyvalent (safeguarding against the "Large Four" snakes of India) in light of his neurotic coagulation profile: krait, saw-scaled snake, Russell's snake, and cobra) counter-agent from Bharat Serums and Immunizations in India, as is customary. After the first hour of taking the antivenom, his priapism subsided and he had complete detumescence. In all, the duration of the priapism was close to three hours. Throughout the next day, he was given an additional 100 mL of antivenom in order to restore normalcy to his coagulation profile. He continued to have regular morning erections free of priapism for the remainder of his hospital stay. He was released from the hospital five days after the snakebite since his condition was stable and his vital signs and coagulation profile were normal. His early morning erections were typical, and he was determined to be stable over the four weeks of weekly follow-up. This patient did not exhibit any symptoms consistent with PTSD or any other mental illness.




| Investigation | Results | Unit | Normal range |
|---|---|---|---|
| Haemoglobin | 14.1 | gms% | 13.0-16.0 |
| Total RBC count | 4.61 | millions/uL | 4.00-5.00 |
| HCT | 42.3 | % | 41.00-50.00 |
| MCV | 91.8 | fl | 81.10-96.00 |
| MCH | 30.6 | Pg | 27.20-33.20 |
| MCHC | 33.3 | % | 32-36 |
| Total WBC count | 19.6 | $x10^3$ cells/uL | 4.00-11.00 |
| Neutrophils | 16.13 | $x10^3$ cells/uL | 2.0-7.0 |
| Lymphocytes | 2.17 | $x10^3$ cells/uL | 1.0-3.0 |
| Monocytes | 1.09 | $x10^3$ cells/uL | 0.1-08 |
| Eosinophils | 0.16 | $x10^3$ cells/uL | 0.02-0.5 |
| Basophils | 0.05 | $x10^3$ cells/uL | 0.02-0.1 |
| Platelet count | 316 | $x10^3$ cells/uL | 150-450 |
| MPV | 9.7 | fl | 6.5-12.0 |

| | | | | |
|---|---|---|---|---|
| PDW | 10.2 | fl | 9.0-13.0 | |
| Prothrombin time | 19.0 | Seconds | 11.5-16.0 | |

**Table 1: Basic Laboratory examination results of the Victim**

HCT, haematocrit; MCH, mean corpuscular haemoglobin; MCHC, m
MCV, mean corpuscular volume; MPV, mean platelet volume; PDW,
WBC, white blood cell.

## CASE DISCUSSION

Priapism, which may be caused by either an increase (nonischaemic) or a reduction (ischaemic) the flow of blood to the penis, is unexpected and infrequently seen in clinical practice [10]. It is clinically probable that one or more components of the venom produced the priapism in this victim if a penile erection develops after envenomation and gradually fades away after antivenom therapy. Clinical examples and experimental models have shown penile erections after exposure to venoms of box jellyfish, widow spiders, and scorpions, and the reasons behind this phenomenon are now being investigation [9]. It seems that nitric oxide synthase (NOS) activation during arachnid envenomation occurs as a result of downstream calcium flow and delayed inactivation of sodium channels in corpus cavernosum tissue [9,11,12]. The poison's effects on certain molecules in the human corpus cavernosa, such as nitric oxide (NO) [13], phosphodiesterases (PDE) [14], ion channels, and N-methyl-D-aspartate (NMDA) [15] receptors, all of which are involved in the regulation of erection, may be the cause of a penile erection after being envenomated by a Russell's viper. It is plausible that the toxins causing this effect are either neutralized in more seasoned snakes or just communicated in the toxin of adolescent snakes, considering that defibrinating compounds like ancrod and batroxobin from different vultures have been proposed to treat priapism in people [17,18] and that toxins from adolescent Russell's vultures show decreased protein articulation [16]. If snake venom's action is mediated by NMDA receptors, then priapism could be caused by an imbalance of polyamines [19]. Hypopituitarism is another unusual in any case, irrefutable confusion of Russell's snake envenomation that influences the sexual organs through pituitary deficiency [20].It is essential to investigate whether these unexpected impacts on the pituitary organ or other organs are connected to the venom's activity via brain synaptosomes, given the neurotoxic impacts of Russell's snake toxin. Except for a couple of poisons, (for example, apamin from honey bee toxin) that have been shown to do so [21,22], it is essential to keep in mind that venom may primarily affect tissues on the periphery because the majority of venom proteins cannot cross the blood-brain barrier [21]. Pregabalin targets voltage-gated calcium channel subunits, leading other researchers to believe that they are the cause of this disease [23].An horse veterinarian case study reveals a potential biological connection between the pituitary pars intermedia and infertility [24]. An article was published on a protein complex in Russell's viper venom that is hazardous to reproduction. The study found that dosed mice showed an increase in NO in their testis, but no priapism [25]. Priapism after Tityus serrulatus envenomation is thought to be caused by NO, which relaxes the corpora cavernosa [26]. Despite the fact that this has not been accounted for in SBE casualties so far, additional research into how venoms affect pathways like NO and ion channels may shed light on female sexual dysfunction because the underlying processes are very similar [27].The underdiagnosis and undertreatment of female sexual dysfunction is a serious problem that requires further study [28]. Studying the effects of toxin poisons in this setting might uncover the pharmacological mechanisms at work and lead to new treatment options for erectile dysfunction in men and women due to the peculiar envenomation effect of Russell's viper bite.

While it is still too soon to say how particular venom toxins affect erectile dysfunction (ED), this unusual occurrence of priapism after a snakebite could lead researchers to look into Russell's viper venom toxins. Since 90% of men over 80 report being unhappy with their sexual performance, there is a large market for erectile dysfunction therapies like PDE5 inhibitors (sildenafil citrate, e.g., viagra, taladafil, and vardenafil)and continuous research into discovering better alternatives. An erection occurs when your blood vessels dilate and your blood flow increases because soluble guanylate cyclase is activated by the release of NO from endothelial cells in response to sexual stimulation. They only work in a small number of patients because they rely on NO-loosening up nerve filaments and corpus enormous endothelium to keep cGMP from breaking down [9, 10]. Thus, alternatives to pharmaceuticals for erectile dysfunction that do not cause unwanted side effects may be available via the use of venom toxins that have developed naturally. In research, P. nigriventer venom-derived toxins have shown promise, and synthetic PnTx2-6 derivatives have eliminated undesirable side effects like pain and cerebral edema. In addition, these compounds may provide an alternate to PDE5 inhibitors by reducing the inactivation rates of voltage-gated Na+ channels (Nav). At this time, the exact mechanisms by which Russell's viper venom activates NaV channels remain unknown. Nevertheless, it is known that some snake venoms include NaV channel activators. It is interesting to note that Russell's snake toxin produces vasodilation through voltage and calcium-set off potassium channels Kv and KCa, so we may conduct additional research on this potential mechanism for cutting-edge treatments for erectile dysfunction. Additionally, the venom of numerous snakes, including the Russell's viper, contains PDEs.Because the venom contains both the venom enzymes and the inhibitors of those enzymes, it may be able

to protect the host against the poison itself. Thus, Russell's viper venom may include both PDEs and PDE inhibitors, the ratio of which may be unbalanced in young snakes.

## CONCLUSION

This kind of unusual event could serve as a springboard for novel investigations into the mechanisms of venom toxins, which might improve our knowledge of the pathophysiology of envenomation. The exact mechanism by which Russell's viper venom induces priapism, as well as the evolutionary advantage of this toxin or if it is a very unusual physiological reaction, remain unknown at this time. Indeed, there are a number of documented conditions that may promote priapism, including sickle cell disease in males, vasculitis, long-term drug usage, trauma (such as intense bicycle riding), and so on. Hence, more studies are needed to ascertain the consequences of priapism-inducing venoms from Russell's vipers. We still don't know much about the molecular connections between priapism and snake venom toxins from this case report, but at least we have data to start a new path of inquiry in this important field. Particularly, since SBE is prevalent in remote tropical regions, doctors need to be trained to recognize atypical consequences like these for diagnostic and research purposes. This article emphasizes that it is necessary to document atypical clinical instances of SBE and SBE-prompted priapism to decide if this impact is unprecedented or well defined for a subset of Russell's snakes. This clinical situation might give new stages to the making of diagnostics, research apparatuses, and therapeutic molecules. It is essential to fully comprehend the consequences of envenomation in order to develop better SBE treatment options.

## REFERENCE

1. Kasturiratne A, Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, Premaratna R,et al. The global burden of snakebite: a literature analysis and modelling based on regional estimates ofenvenoming and deaths. PLoS Med. 2008; 5(11):e218. https://doi.org/10.1371/journal.pmed.0050218 PMID: 18986210

2. Chippaux JP. Snake-bites: appraisal of the global situation. Bull World Health Organ. 1998; 76(5):515–24. PMID: 9868843

3. Williams HF, Layfield HJ, Vallance T, Patel K, Bicknell AB, Trim SA, et al. The Urgent Need to DevelopNovel Strategies for the Diagnosis and Treatment of Snakebites. Toxins (Basel). 2019; 11(6). https://doi.org/10.3390/toxins11060363 PMID: 31226842

4. Gutie´ rrez JM, Calvete JJ, Habib AG, Harrison RA, Williams DJ, Warrell DA. Snakebite envenoming.Nat Rev Dis Primers. 2017; 3:17063. https://doi.org/10.1038/nrdp.2017.63 PMID: 28905944

5. Williams SS, Wijesinghe CA, Jayamanne SF, Buckley NA, Dawson AH, Lalloo DG, et al. Delayed psychologicalmorbidity associated with snakebite envenoming. PLoS Negl Trop Dis. 2011; 5(8):e1255.https://doi.org/10.1371/journal.pntd.0001255 PMID: 21829741

6. Leite KR, Andrade E, Ramos AT, Magnoli FC, Srougi M, Troncone LR. Phoneutria nigriventer spidertoxin Tx2-6 causes priapism and death: a histopathological investigation in mice. Toxicon. 2012; 60(5):797–801. https://doi.org/10.1016/j.toxicon.2012.06.006 PMID: 22750220

7. Goel SC, Yabrodi M, Fortenberry J. Recognition and successful treatment of priapism and suspectedblack widow spider bite with antivenin. Pediatr Emerg Care. 2014;30(10):723–4. https://doi.org/10.1097/PEC.0000000000000235 PMID: 25275351

8. Nickson CP, Currie BJ, Fenner PJ. Priapism and Irukandji syndrome. Ann Emerg Med. 2010; 55 (6):581–2. https://doi.org/10.1016/j.annemergmed.2010.01.006 PMID: 20494230

9. Nunes KP, Torres FS, Borges MH, Matavel A, Pimenta AM, De Lima ME. New insights on arthropodtoxins that potentiate erectile function. Toxicon. 2013; 69:152–9.https://doi.org/10.1016/j.toxicon.2013.03.017 PMID: 23583324

10. Gratzke C, Angulo J, Chitaley K, Dai YT, Kim NN, Paick JS, et al. Anatomy, physiology, and pathophysiologyof erectile dysfunction. J Sex Med. 2010; 7(1 Pt 2):445–75. https://doi.org/10.1111/j.1743-6109.2009.01624.x PMID: 20092448

11. Nunes KP, Cordeiro MN, Richardson M, Borges MN, Diniz SO, Cardoso VN, et al. Nitric oxide-inducedvasorelaxation in response to PnTx2-6 toxin from Phoneutria nigriventer spider in rat cavernosal tissue.J Sex Med. 2010; 7(12):3879–88. https://doi.org/10.1111/j.1743-6109.2010.01978.x PMID: 20722794

12. Yonamine CM, Troncone LR, Camillo MA. Blockade of neuronal nitric oxide synthase abolishes thetoxic effects of Tx2-5, a lethal Phoneutria nigriventer spider toxin. Toxicon. 2004; 44(2):169–72. https://doi.org/10.1016/j.toxicon.2004.05.016 PMID: 15246765

13. Xavier FE. Nitrergic perivascular innervation in health and diseases: Focus on vascular tone regulation.Acta Physiol (Oxf). 2020; 230(1):e13484. https://doi.org/10.1111/apha.13484 PMID: 32336027

14. Halle´n K, Wiklund NP, Gustafsson LE. Inhibitors of phosphodiesterase 5 (PDE 5) inhibit the nerveinducedrelease of nitric oxide from the rabbit corpus cavernosum. Br J Pharmacol. 2007; 150(3):353–60. https://doi.org/10.1038/sj.bjp.0706991 PMID: 17179943

15. Staudt MD, de Oliveira CV, Lehman MN, McKenna

KE, Coolen LM. Activation of NMDA receptors inlumbar spinothalamic cells is required for ejaculation. J Sex Med. 2011; 8(4):1015–26. https://doi.org/10.1111/j.1743-6109.2010.02168.x PMID: 21235717

16. Tun P, Nu Nu L, Aye Aye M, Kyi May H, Khin Aung C. Biochemical and biological properties of thevenom from Russell's viper (Daboia russelli siamensis) of varying ages. Toxicon. 1995; 33(6):817–21.https://doi.org/10.1016/0041-0101(95)00032-h PMID: 7676473

17. Bell WR Jr. Defibrinogenating enzymes. Drugs. 1997; 54 Suppl 3:18–30; Discussion -1. https://doi.org/10.2165/00003495-199700543-00005 PMID: 9360849

18. Bell WR, Pitney WR. Management of priapism by therapeutic defibrination. N Engl J Med. 1969; 280(12):649–50. https://doi.org/10.1056/NEJM196903202801207 PMID: 5764844

19. Aird SD, Villar Briones A, Roy MC, Mikheyev AS. Polyamines as Snake Toxins and Their ProbablePharmacological Functions in Envenomation. Toxins (Basel). 2016; 8(10). https://doi.org/10.3390/toxins8100279 PMID: 27681740

20. Antonypillai CN, Wass JA, Warrell DA, Rajaratnam HN. Hypopituitarism following envenoming by Russell'svipers (Daboia siamensis and D. russelii) resembling Sheehan's syndrome: first case report fromSri Lanka, a review of the literature and recommendations for endocrine management. QJM. 2011; 104(2):97–108. https://doi.org/10.1093/qjmed/hcq214 PMID: 21115460

21. Osipov A, Utkin Y. Effects of snake venom polypeptides on central nervous system. Cent Nerv SystAgents Med Chem. 2012; 12(4):315–28. https://doi.org/10.2174/187152412803760618 PMID:23270323

22. Oller-Salvia B, Teixido´ M, Giralt E. From venoms to BBB shuttles: Synthesis and blood-brain barriertransport assessment of apamin and a nontoxic analog. Biopolymers. 2013; 100(6):675–86. https://doi.org/10.1002/bip.22257 PMID: 24281722

23. Alsulihem AA, Rabah DM. Priapism associated with pregabalin. Urol Ann. 2014; 6(4):366–8. https://doi.org/10.4103/0974-7796.141012 PMID: 25371619

24. Shepard MK, Lee WL, Eggleston RB. Perianesthetic development of diaphragmatic hernia in a horsewith equine pituitary pars intermedia dysfunction (PPID). Can Vet J. 2015; 56(1):48–52. PMID:25565714

25. Kumar JR, Basavarajappa BS, Arancio O, Aranha I, Gangadhara NS, Yajurvedi HN, et al. Isolation andcharacterization of "Reprotoxin", a novel protein complex from Daboia russelii snake venom. Biochimie. 2008; 90(10):1545–59. https://doi.org/10.1016/j.biochi.2008.05.018 PMID: 18573307

26. Teixeira CE, de Oliveira JF, Baracat JS, Priviero FB, Okuyama CE, Rodrigues Netto N Jr., et al. Nitricoxide release from human corpus cavernosum induced by a purified scorpion toxin. Urology. 2004; 63(1):184–9. https://doi.org/10.1016/s0090-4295(03)00785-4 PMID: 14751389

27. Musicki B, Liu T, Lagoda GA, Bivalacqua TJ, Strong TD, Burnett AL. Endothelial nitric oxide synthaseregulation in female genital tract structures. J Sex Med. 2009; 6 Suppl 3(S3proceedings):247–53.https://doi.org/10.1111/j.1743-6109.2008.01122.x PMID: 19138376

28. Clayton AH, Valladares Juarez EM. Female Sexual Dysfunction. Psychiatr Clin North Am. 2017; 40 (2):267–84. https://doi.org/10.1016/j.psc.2017.01.004 PMID: 28477652.