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Case report

Russell's viper envenomation induces rectus sheath haematoma

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ABSTRACT

Snakebite envenomation causes systemic and local manifestations, which result from the individual or synergistic actions of multiple venom components. The pathological hallmarks of medically important venomous snakes such as the Indian Russell's viper (*Daboia russelii*) are well known. Envenomation by Russell's viper is typically characterised by coagulopathies, muscular damage, nephrotoxicity, and neurotoxicity. However, recent reports have revealed several unusual complications that provide a better understanding of Russell's viper envenomation effects. To further strengthen this, here, we report a case of Russell's viper bite that induced acute abdominal pain, which was intensified on day two and conservatively treated under medical supervision. Both Fothergill and Carnett signs were positive for this patient. An ultrasound imaging revealed a dissimilar dense mass, and the abdominal computed tomography scan confirmed rectus sheath haematoma. The clinical management involved the administration of polyvalent antivenom, packed red blood cells, fresh frozen plasma, and platelets. The patient recovered gradually and was discharged from the hospital eight days after the bite. Overall, this case presentation shares an uncommon experience and adds new insights into the complex series of rare pathological events associated with Russell's viper bites in India. The scientific documentation of relatively infrequent entities based on an ongoing living assessment of medical experiences, for example, this rectus sheath haematoma, constitutes valuable guidance for an adequate diagnosis and timely treatment. Essential awareness among clinicians and further research on understanding the molecular relationship between Russell's viper venom and rectus sheath haematoma will improve patient outcomes and understanding of this condition, respectively.

1. Introduction

Snakebite envenomation (SBE), a high-priority neglected tropical disease, is associated with a range of clinical conditions leading to deaths or permanent disabilities (Gutiérrez et al., 2017). Successful clinical management of SBE requires prompt diagnosis, adequate medical training for healthcare professionals, and a deeper understanding of the broad spectrum of clinical manifestations including rare complications (Hamza et al., 2021). India remains the 'capital of SBE' due to a large number of incidents resulting in more than 58,000 deaths every year (Suraweera et al., 2020). Russell's viper (*Daboia russelii*) is the protagonist snake involved in the majority of SBE in India, accounting for over 40% of incidents (Vaiyapuri et al., 2013; Samuel et al., 2020; Senji Laxme et al., 2021). The classic symptoms of Russell's viper envenomation are coagulopathy, local muscle damage, nephrotoxicity, and neurotoxicity (Mahasandana et al., 1980; Kumar et al., 2018; Ratnayake et al., 2019). However, several recently published articles have highlighted that the mode of action and clinical complications caused by the Indian Russell's viper venom are much broader. For example, the Indian Russell's viper induced several rare complications such as the Wunderlich syndrome (Senthilkumaran et al., 2022a,b), pseudoaneurysm (Senthilkumaran et al., 2022a,b), priapism (Senthilkumaran et al., 2021) and salivary calculus development in submandibular gland (Arathisenthil et al., 2022). Similarly, here, we report a case of rectus

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sheath haematoma (RSH) in a female patient following Russell's viper envenomation in India.

RSH is an uncommon, and underdiagnosed disorder characterised by an accumulation of blood in the anterior rectus abdominis muscle (Paschou et al., 2014, Mahamad Arif and Syed Alwee Al'Aidrus, 2021). The pathogenesis of RSH is multifactorial with non-specific nature of clinical signs and symptoms. Its manifestations are varied depending on several factors, for example, the degree of peritoneal irritation, and the extensivity and location of the haematoma (Hatjipetrou et al., 2015). As an acute abdominal presentation, RSH is often accompanied by severe/sharp non-radiating pain, vomiting, tenderness, fever, chills, and nausea. Notably, a lower palpable abdominal mass that never crosses the midline is a typical feature of the RSH (Siu et al., 2003). RSH is usually misdiagnosed and easily confused with more prevalent intra-abdominal conditions, i.e. appendicitis, incarcerated inguinal hernia, and cholecystitis, among others (Buffone et al., 2015). Fothergill and Carnett signs are clinical clues that can guide and assist in reaching an earlier diagnosis of RSH (Yale et al., 2020). The non-invasive computed tomography scanning constitutes a gold standard method for identifying and monitoring this condition (Bello and Blanco 2019). To the best of our knowledge, the development of RSH following SBE has not been previously described in the literature. Hence, this report provides essential awareness and treatment guidelines for clinicians specifically in rural areas to tackle this issue following envenomation by Russell's viper and other snakes.

2. Case report

A 46-year-old female without any comorbidities was bitten by a snake on her left foot while she was working in a sugarcane field. The offending snake (which was brought to the hospital) was identified as Russell's viper by a trained herpetologist (Fig. 1A). The patient was taken to a local hospital immediately (within 30 minutes) after the bite, where she received 100 mL (10 vials) of polyvalent antivenom raised against the Indian 'Big Four' snakes (Russell's viper, Indian cobra, krait, and saw-scaled viper) as she demonstrated pain, local oedema and haematological [prolonged 20 minutes whole blood clotting test and international normalised ratio (INR) of blood clotting] manifestations, typical clinical features of Russell's viper envenomation. The administration of antivenom has corrected these manifestations, although, on the second day following the bite, she developed sudden sharp abdominal pain associated with nausea but there was no history of vomiting, loose stools, or fever. An ultrasound examination of the abdomen confirmed a non-mobile heterogeneous dense mass on the left upper quadrant extending along the omentum. The pain lasted for the whole day and gradually worsened with a significant drop in haemoglobin levels from 9 g/dL to 5 g/dL. Hence, she was referred (around 24 hours after the bite) to the emergency department of our hospital for further management.

Upon arrival, she was conscious but anxious and looked pale and afebrile. Her blood pressure was 80/60 mmHg with a heart rate of 122 beats/min and room air saturation of 92%. She had abdominal tenderness at the lower left quadrant of the abdomen and a palpable mass. The mass was tender on palpation, but there was no rebound tenderness and muscular rigidity. Both Fothergill and Carnett tests (clinical tests to ascertain RSH in patients with any abdominal injuries) were positive. Auscultation of the abdomen revealed a hypoactive bowel sound. Her haemoglobin level was 4.0 g/dL with a haematocrit of 12.8% and the platelet count was 86,000/μl (Table 1). The activated partial thromboplastin time (aPTT) was 41.8 seconds, prothrombin time (PT) was 20.7 seconds, and the INR of clotting was 2.1. All other biochemical tests were normal (Table 1). The ultrasound scan was performed to rule out any intra-abdominal haemorrhage, and it confirmed no solid organ injury or free fluid. Therefore, a CT scan of the abdomen was performed, and it revealed a haematoma on the rectus sheath of the abdominal wall (Fig. 1B). There was no predisposing condition in this patient including anti-coagulation therapy, constipation, coughing and family history of bleeding diathesis or haematological diseases. There were also no renal impairments in this victim. She received 10 vials (100 mL) of polyvalent antivenom raised against the Indian 'Big Four' snakes (Bharath Serums and Vaccines Limited, Mumbai, India) over 45 minutes in 250 mL of normal saline. In addition, six units each of packed red blood cells, fresh frozen plasma, and platelet concentrates were administered. The abdominal pain gradually subsided, her haemodynamics were optimised, haemoglobin values increased to 9.5 g/dL and the INR value decreased to 1.27. She was discharged eight days later (from admission to our hospital). The weekly follow-up monitoring over three months did not reveal any further effects in this patient.

3. Discussion

SBE-induced abdominal bleeding is an infrequent event that has been poorly documented in the literature. Recently, a rare case of retroperitoneal and peritoneal diffuse haematomas in a 11-year-old child

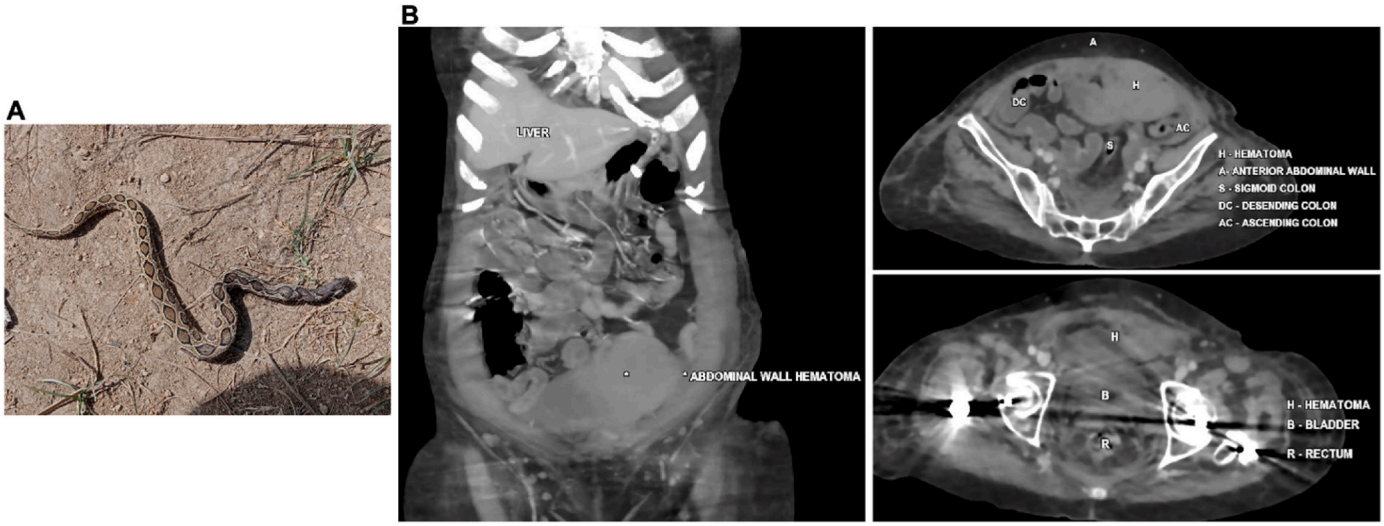

Fig. 1. The development of rectus sheath haematoma in a patient following Russell's viper bite. A, the offending snake was identified as Russell's viper by a trained herpetologist. **B**, the computed tomography scan images of different views of the abdominal region confirm the presence of a large haematoma at the abdominal wall ascertaining the diagnosis of rectus sheath haematoma.

Table 1
Laboratory results of the patient upon admission to the emergency department.

Specimen	Investigation	Results	Unit	Normal range
EDTA Whole Blood	Haemoglobin	4.0	gms%	13.0–16.0
EDTA Whole Blood	Total RBC count	3.45	Millions/μL	4.00–5.00
EDTA Whole Blood	HCT	12.8	%	41.00–50.00
EDTA Whole Blood	MCV	88.1	fl	81.10–96.00
EDTA Whole Blood	MCH	29.0	pg	27.20–33.20
EDTA Whole Blood	MCHC	32.9	%	32–36
EDTA Whole Blood	Total WBC count	16.97	$x10^3$ Cells/μL	4.00–11.00
EDTA Whole Blood	Neutrophils	14.8	$x10^3$ Cells/μL	2.0 to 7.0
EDTA Whole Blood	Lymphocytes	0.79	$x10^3$ Cells/μL	1.0 to 3.0
EDTA Whole Blood	Monocytes	1.36	$x10^3$ Cells/μL	0.1 to 0.8
EDTA Whole Blood	Eosinophils	0	$x10^3$ Cells/μL	0.02 to 0.5
EDTA Whole Blood	Basophils	0.02	$x10^3$ Cells/μL	0.02 to 0.1
EDTA Whole Blood	Neutrophils	87.2	%	55–75
EDTA Whole Blood	Lymphocytes	4.7	%	15–30
EDTA Whole Blood	Eosinophils	0	%	1–5
EDTA Whole Blood	Monocytes	8.0	%	2–10
EDTA Whole Blood	Basophils	0.1	%	Up to 1
EDTA Whole Blood	Platelet Count	86	$x10^3$ Cells/μL	150–450
EDTA Whole Blood	MPV	9.4	fl	6.5–12.0
EDTA Whole Blood	PDW	8.4	fl	9.0–13.0
Serum	Urea	33	mg/dL	15–40
Serum	Creatinine	0.98	mg/dL	0.6–1.2
Serum	Uric acid	3.5	mg/dL	2.4–6.1
Serum	Bilirubin (total)	1.10	mg/dL	0.2–1.2
Serum	Bilirubin (direct)	0.37	mg/dL	0–0.2
Serum	Bilirubin (indirect)	0.73	mg/dL	0.2–0.9
Serum	SGOT	32	U/L	5–35
Serum	SGPT	40	U/L	5–45
Citrated plasma	Prothrombin time	20.7	Seconds	11.5–16.0
Citrated plasma	aPTT	41.8	Seconds	26.0–40.0
Citrated plasma	INR	2.1	Ratio	

RBC, red blood cells; HCT, haematocrit; MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; WBC, white blood cells; MPV, mean platelet volume; PDW, platelet distribution width; aPTT, activated partial thromboplastin time; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamate-pyruvic transaminase.

following a severe envenomation was reported (Kassegne et al., 2020). So far, RSH is an under-recognised cause of acute abdominal pain in SBE victims. To the best of our knowledge, the relationship between RSH and SBE has not been previously reported. In general, RSH occurs due to the tearing of the epigastric arteries or damage to intramuscular blood vessels because of tearing/damage to the muscle itself. Based on a retrospective analysis of the characteristics of the patients, this devastating bleeding complication is usually located infraumbilically and never crosses the midline, barring a few exceptions (Sabanis et al., 2020). Nevertheless, haematoma below the level of the arcuate line of Douglas causes an indirect irritation on the peritoneum due to the absent posterior rectus sheath in this region masquerading as acute abdomen pain (Kapan et al., 2008). Multiple etiological factors have been identified for RSH. Usually, it has been linked to trauma, abdominal surgery, trocar site injury after laparoscopic surgeries, subcutaneous drug injections, warfarin therapy, haematological diseases, uncontrolled hypertension, violent coughing, vigorous physical exercise, and pregnancy. However, in a few rare cases, it also developed spontaneously (Nizam et al., 2020). In this study, we report the development of RSH in a 46-year-old female victim following Russell's viper bite and clinical management strategies used for the diagnosis and treatment of this condition. This patient developed RSH despite prompt antivenom treatment in a local hospital following the bite.

The probable mechanism of RSH in the patient reported in this study may be due to a combination of thrombocytopenia, increased vascular fragility, dysfunctional platelets, coagulopathy, and increased fibrinolysis (Slagboom et al., 2017). From a pathogenesis standpoint, these haemodynamic disturbances are probably orchestrated by toxins that damage the endothelial layer of the vasculature (Rucavado et al., 2018), procoagulant and fibrinolytic enzymes (Mukherjee 2014), and proteins that modulate platelet function (Kalita et al., 2019). Factor X activator (RVV-X) (Takeya et al., 1992), factor V activator (RVV-V) (Tokunaga et al., 1988), daborhagin-M and daborhagin-K (Chen et al., 2008) are classical examples of toxins in Russell's viper venom related to haemostatic disturbances. Earlier studies have demonstrated that Russell's viper venom components trigger multifocal toxicity through dynamic changes in the levels of coagulation factors accompanied by consumptive coagulopathy and severe impact on clotting cascades (Isbister et al., 2015). On the other hand, snake venom metalloproteases (SVMPs) exert their haemorrhagic effects by cleaving key components of the basement membrane of capillaries with a direct impact on the mechanical stability of vessel walls (Gutiérrez et al., 2016a). The understanding of this biological phenomenon was built based on experimental results obtained using microscopic, histological, biochemical, and proteomic strategies (Gutiérrez et al., 2016b). Therefore, clinical findings, such as uncommon RSH, add a new dimension to the current views of SBE-induced bleeding complications that must be addressed through future research using integrative approaches including prospective clinical studies, omic tools and *in vitro, ex vivo* and/or *in vivo* functional assays to establish the molecular interactions between venom toxins and key factors that regulate the cardiovascular system, specifically blood coagulation.

Diagnosis of RSH can be challenging, mainly because its unspecific symptoms are highly similar and often shared with other causes of acute abdominal pain (Costello and Wright 2005). As reported in this patient, overlapping clinical manifestations must be carefully evaluated to avoid misdiagnosis or delayed diagnosis, which may hinder early and non-invasive intervention. The ability to recognise important clinical signs prevents negative exploratory laparotomies and consequently reduces morbidity and mortality risks associated with surgical procedures. Fothergill and Carnett signs were positive in this SBE victim, similar to clinical examination among patients with different aetiologies of the RSH (Yale et al., 2020). These physical findings are fundamental elements of the diagnostic process, contributing to the differentiation of RSH from other frequent intra-abdominal conditions. The basic principle behind these bedside techniques is the assessment of the position of a palpable mass and the modulation of pain intensity in response to tensing of the abdominal muscles (Yale et., 2020). The presence of an unchanged abdominal mass in terms of location (does not cross the midline), which can be palpated in both relaxed and contracted muscle conditions, characterises Fothergill sign (Bello and Blanco 2019). The other uncomplicated test of high diagnostic value called Carnett sign is associated with increased or maintained pain and tenderness during muscle movement. To illustrate this point, as a first step, clinicians delimit the region of maximum tenderness by palpation in the resting state. Then, the patient tenses the abdominal wall by lifting the head and shoulders or both legs, while the site of pain continues to be monitored. Confirmation of the sign considers the comparison of pain and

tenderness in both circumstances. In summary, this test is considered positive when the pain during contraction is equal to or more intense than the initial screening (Yale et., 2020). These methods were reported and proposed for the recognition of abdominal wall haematoma approximately a century ago and it remains useful to this day (Yale et., 2020). Hence, awareness of this rare clinical entity and its symptoms is crucial in the differential diagnosis of acute abdominal pain and in the development of an alternative and cost-effective strategy in under-resourced rural settings. These aspects must be considered due to the disproportionate impact of SBE in tropical rural areas with under-resourced health systems (Gutiérrez et al., 2017). Taken together, the simple clinical criteria discussed above may assist in better triage of patients with RSH, including those caused by SBE (Senthilkumaran et al., 2012). In the same context, the combination of diagnostic imaging techniques (e.g., ultrasonography and CT scans) will provide a significant increase in sensitivity and specificity in the diagnosis of acute abdominal pain (Pierro et al., 2018). Ultrasonography is a valuable imaging tool, however, it plays a relatively limited role in the diagnosis of RSH. This approach is non-specific with a 71% sensitivity (Moreno Gallego et al., 1997). Occasionally, it is difficult to differentiate intra-peritoneal lesions from extra-peritoneal lesions by ultrasound, as seen in our case. The limitations rely on the interpretation of the images and errors derived from the probe-induced tenderness (Costello and Wright 2005). However, CT scans produce high-quality images and show better discriminative ability in terms of localisation, size, origin, extension, and evaluation of the collection of blood outside of the blood vessels in the rectus sheath (Hamza et al., 2021). Thus, CT evaluation aids in narrowing down the correct diagnosis and it should be adopted as a first-line technique when available.

Prompt clinical management remains a cornerstone in the treatment of haemodynamically stable patients with no evidence of active bleeding or expanding haematoma (Liao and Puckett 2021). In our patient, the clinical outcomes were favourable following primary treatment with antivenom, packed cells, fresh frozen plasma, and platelet transfusion. This first-line treatment is reported as an appropriate choice for stable individuals that do not require surgical exploration. Similar combinatory therapy has been successfully employed in other RSH cases in non-SBE patients (Kapan et al., 2008; Buffone et al., 2015). Invasive control of active bleeding is considered only if conservative treatments fail and the clinical severity criteria characterised by haemodynamic instability, neurological deficit and continued bleeding are present (Pierro et al., 2018). In line with this, surgical intervention was considered unnecessary for this patient due to positive clinical evaluation and possible complications associated with this aggressive procedure. In addition, postoperative mortality after RSH surgical management is relatively high with the risk of re-bleeding (Cereda et al., 2017). Therefore, clinicians have employed endovascular coil embolisation, as an alternative in those patients who are refractory to conservative treatment that requires additional intervention (Senthilkumaran et al., 2009). This minimally invasive therapeutic modality has ensured high clinical success with recognised safety, effectiveness and low morbidity (Méndez et al., 2022).

In conclusion, the conservative diagnosis and treatment based on the recognition of simple signs and imaging modalities proved to be very useful for a successful outcome in this patient with RSH. The improvement of the clinical management for SBE requires multidisciplinary strategies, including robust training and knowledge about the unusual manifestations, their diagnosis, and therapies. The detailed description of rare clinical observations such as RSH following Russell's viper bites represents a starting point that opens avenues for further studies from basic research towards the clinic and helps to establish standard treatment protocols for SBE.

Credit author statement

Subramanian Senthilkumaran: Conceptualization, Methodology, Validation, Investigation, Resources, Data curation, Visualization, Supervision. **José R. Almeida:** Writing – original draft, Writing – review & editing, Visualization. **Jarred Williams:** Writing – original draft, Writing – review & editing, Visualization. **Anika Salim:** Writing – original draft, Writing – review & editing, Visualization. **Harry F. Williams:** Formal analysis, Writing – review & editing, Supervision. **Ponniah Thirumalaikolundusubramanian:** Formal analysis, Validation, Investigation, Resources, Data curation, Writing – original draft, Writing – review & editing, Supervision. **Ketan Patel:** Formal analysis, Resources, Writing – review & editing. **Sakthivel Vaiyapuri:** Conceptualization, Methodology, Formal analysis, Validation, Investigation, Resources, Data curation, Writing – original draft, Writing – review & editing, Visualization, Supervision.

Ethical statement

This research was conducted according to the Declaration of Helsinki and the ethical guidelines of the Indian Council of Medical Research. The data collection, consent form, and information sheet were approved by the Institutional Ethics Committee at Toxiven Biotech, Tamil Nadu, India (Reference number: ICMR-Toxiven Ethics, 2022/1) and University of Reading Research Ethics Committee (Reference number: UREC 23/05). A written consent was obtained before this case report was published.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

All the data associated with this article are presented within this article.

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