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Abstract

Splenic rupture and/or splenectomy is/are not uncommon in clinical arena. Here we present this case of extensive haemorrhage-induced splenic rupture which resulted in splenectomy in a young healthy male (who did not have any previous medical conditions) following a Russell's viper bite. He developed upper abdominal and shoulder pain on his left side along with hypotension and reduced level of haemoglobin on the third day following bite despite antivenom treatment. Following confirmation of splenic rupture and haemoperitoneum by ultrasound and computed tomography scans, an emergency splenectomy was performed using laparotomy. Although Russell's viper bites are known to induce bleeding complications, splenic rupture due to haemorrhage in spleen has not been previously reported. Russell's viper venom toxins such as metalloproteases, serine proteases and phospholipase A_2 might have affected the vascular permeability resulting in excessive bleeding and increased pressure in the spleen leading to rupture. Further investigations are required to underpin the impact of snake venom toxins on the architecture and functions of spleen. However, the clinicians who treat snakebites should be aware of this type of rare complications so as to provide appropriate management for such victims.

Key words

Snakebite envenomation; Russell's viper; splenic haemorrhage; non-traumatic splenic rupture; haemoperitoneum; splenectomy

Introduction

Snakebite envenomation (SBE) has been classified as a high priority neglected tropical disease by the World Health Organisation (Chippaux, 2017). SBE-induced deaths, disabilities and socioeconomic ramifications are more prevalent among the rural communities living in developing countries (Kasturiratne et al., 2008; Vaiyapuri et al., 2013; Williams et al., 2019). Snake venoms are a blend of enzymatic and non-enzymatic proteins/peptides that induce a wide range of envenomation effects including haemotoxic, myotoxic, cytotoxic, nephrotoxic, and neurotoxic complications (Williams et al., 2019). The elapid and viper snakes are responsible for a majority of lethal SBE cases. While elapid bites predominantly induce neurotoxic effects, the bites from viper snakes largely display haemotoxic effects (Gutiérrez et al., 2017; Williams et al., 2019). Russell's viper (*Daboia russelii*) is one

of the critical species of Indian 'Big Four' snakes and responsible for most bites in India (Suraweera et al., 2020; Vaiyapuri et al., 2013). The bites from Russell's vipers are unique as they exhibit mostly haemotoxic effects along with specific neurotoxic complications (Warrell, 1989). Splenic rupture is often caused by trauma due to the delicate nature of this organ. However, it can also occur without traumatic injury which is known as atraumatic, non-traumatic or spontaneous splenic rupture less frequently due to various causes (Lieberman and Levitt, 1989). Multiple systematic analyses were performed on the causes of atraumatic splenic rupture, however, SBE (specifically Russell's viper bite) has not been previously reported as a causative reason (Lieberman and Levitt, 1989; Renzulli et al., 2009) although few cases of splenic rupture following SBE have been reported (Kang et al., 2014; Kim et al., 2021; Lee and Sung, 2019; Yhi et al., 2013). A single case reported in the 19th century demonstrates a cobra bite leading to congestion in all organs including spleen (reviewed in (Feola et al., 2020)). Similarly, few case studies have reported splenic rupture following SBE from unidentified snake species in South Korea (Kang et al., 2014; Kim et al., 2021; Lee and Sung, 2019; Yhi et al., 2013). Moreover, spontaneous splenic rupture due to antivenom treatment has not been reported either. Hence, we report this unique case of a Russell's viper (from Tamil Nadu, Southern India) bite resulting in splenic rupture and subsequent splenectomy to demonstrate such unusual complications of SBE even after administration of antivenom. We believe that this will create awareness among the practitioners and emergency physicians about non-traumatic splenic rupture in SBE victims.

Case presentation

A 30-year-old healthy male with no previous history of any medical conditions was admitted in a local hospital within two hours following a snakebite on his right great toe while he was harvesting sugarcane in his field. The offending snake was killed and brought to the hospital and identified as a Russell's viper by an expert herpetologist (**Figure 1A**). The patient complained of severe pain over his right foot and the local examination confirmed two fang marks with reddish edema. No other haemorrhagic or neurological manifestations were observed clinically upon admission. His 20-minute whole blood clotting time test (20-WBCT) was prolonged and therefore, he received continuous intravenous administration of 150 mL polyvalent antivenom (within two and half hours following bite) to normalise his clotting time. His haematologic, biochemical and coagulation parameters were starting to improve after antivenom treatment. For example, his 20-WBCT was getting improved in a stepwise manner over time: at admission (0 hour) - prolonged; 6 hours - 25 minutes; 12 hours - 22 minutes; 24 hours - 14 minutes; 36 hours - 8 minutes; 48 hours - 8 minutes.

However, on the third day of hospitalisation, he developed sudden onset of severe pain on his left upper abdominal area and shoulder along with nausea. His 20-WBCT was 24 minutes at 54 hours. The pain was constant, and it did not reduce with standard pain management drugs. Hence, he was referred to our emergency department (ED). Local examination established swelling and a focal haemorrhagic bulla at the bite site (**Figure 1B**). Moreover, he was drowsy, disoriented and appeared pale but not cyanosed or jaundiced. He was afebrile; tachypnoeic with a heart rate of 140 beats per minute (tachycardia) in sinus rhythm, and blood pressure of 60/40 mmHg with room air oxygen saturation of 90%. On pulmonary auscultation, bilaterally equal breathing sound without any added sounds was noted but his abdomen was distended with tenderness in left hypochondrium. There was no obvious systemic or subcutaneous haemorrhage, bleeding from gums, or purpura. His abdominal ultrasonography revealed intraperitoneal fluid collection in Morison's pouch, the splenorenal recess, and supra pubic space. Contrast enhanced computed tomography (CT) scan of abdomen showed a gross haemoperitoneum with splenic rupture (**Figure 1C**). The patient and his family members denied any known history of thoracoabdominal trauma. A significant reduction in his haemoglobin level from 14 g/dL upon admission at the previous hospital to 6.0 g/dL in our ED was observed. His white blood cell (WBC) count was 16,400/µL, and platelet count was 189,000/µL in our laboratory investigation (**Table 1**). Serum electrolytes and renal function tests were normal. Nevertheless, his 20-WBCT, prothrombin/ international normalised ratio of clotting and activated partial thromboplastin times were prolonged. Therefore, 100 mL of antivenom (Bharat Serums and Vaccines Limited, India) was administered intravenously to normalise his coagulation abnormalities. He also received paracetamol infusion (1 g over 12 hours) to manage his pain in the ED. Moreover, he was transfused with four units each of fresh

frozen plasma, platelet concentrate and packed cells to normalise his coagulation status. Despite these interventions, his haemodynamic parameters did not improve. Hence, an exploratory laparotomy was performed, which identified ruptured subcapsular splenic haematoma as well as lacerated and traumatised spleen. This investigation led to an emergency splenectomy after 12 hours of presentation to our ED. During this procedure, nearly 2200 mL of blood along with blood clots were removed from the abdominal cavity. There was no evidence for haematoma or bleeding from retroperitoneal and perisplenic regions or from any other internal organs. These findings suggest that the spleen is likely to be the primary site for excessive haemorrhage. Intravenous paracetamol was used to control his pain during postoperative period. The external surface of removed spleen displayed haemorrhagic areas at inferior border (**Figure 1D**). The pathological examination revealed thickened capsule with trabeculae arising from the capsule. Significant level of congestion was found in the red pulp although the white pulp appeared to be normal. Hilum showed extensive areas of haemorrhage although there was no evidence for thrombi formation, infarction, and gamma gandy bodies (**Figure 1E**). His postoperative period was uneventful, and the pain was completely reduced. Seven days after splenectomy (i.e. ten days after bite), he was discharged without any complications. Subsequent routine examinations did not show any further abnormalities in this patient.

Case discussion

There are multiple causes for atraumatic or spontaneous splenic rupture such as microbial infection, tumour growth, hyperplasia of splenic cells, physical activities such as weightlifting and rarely, some physiological processes including pregnancy (Halliday et al., 2020; Kaniappan et al., 2018; Lam et al., 2014; Lieberman and Levitt, 1989; Renzulli et al., 2009; Rueda-Esteban et al., 2020). However, the adverse effects of SBE on spleen are rarely encountered. Prior to this study, a cobra bite-induced splenic congestion was observed in a victim in 19th century (as reviewed in (Feola et al., 2020)). Similarly, a total of four cases of splenic rupture following bites from unidentified snake species one to five days after receiving antivenom were reported from South Korea (Kang et al., 2014; Kim et al., 2021; Lee and Sung, 2019; Yhi et al., 2013). Among these cases, three underwent splenectomy (Kang et al., 2014; Kim et al., 2021; Yhi et al., 2013) and in one case, it was averted by successful angioembolisation of splenic artery (Lee and Sung, 2019). To our knowledge, splenic rupture and subsequent splenectomy in Russell's viper bite victims has not been previously reported. Moreover, splenic rupture in a SBE victim due to excessive haemorrhage following antivenom treatment is an unusual clinical event. Hence, we report this case to highlight this rare complication following a Russell's viper bite in India.

The spleen is a highly vascular lymphatic organ that functions primarily as a blood filter and secondarily as a site for initiating immune responses (Cesta, 2006). Its structure comprises the main organ enclosed by connective tissues forming an outer layer or capsule. The delicate nature of this organ with manifold vasculature makes it susceptible for damage. Prominent features of spontaneous or atraumatic splenic rupture include left upper abdominal pain or a distended abdomen along with haemodynamic instability (Renzulli et al., 2009). However, these may not be always helpful in ascertaining splenic rupture. In this case, although the patient has presented these classical symptoms, he was subjected to ultrasound and CT scans to ascertain the splenic rupture prior to splenectomy. Thus, it could be developed as a standard practice to use appropriate scans including easily available and cheaper ultrasound scan to confirm the splenic rupture instead of only relying on the symptoms. Similar to this present case, another SBE incident from South Korea has reported the development of abdominal pain, disorientation, drowsiness, and nausea two days after antivenom treatment (Lee and Sung, 2019). Tachycardia and hypotension along with a significant reduction in haemoglobin level observed in this case closely matches with the symptoms experienced by a SBE victim reported from South Korea (Kim et al., 2021). Other common symptoms observed in our case were also similar to the cases reported earlier (Kang et al., 2014; Kim et al., 2021; Lee and Sung, 2019; Yhi et al., 2013). The abdominal distension and CT findings of gross haemoperitoneum have indicated that bleeding might have occurred within spleen and subcapsular area resulting in increased intrasplenic pressure and splenic rupture. Indeed, the pathological analysis of spleen samples post splenectomy confirmed subcapsular haemorrhage as well as congestion and haemorrhage in the red pulp of the spleen. The patient in this case appears to have triggered a high level of immune response as indicated by the

elevated levels of white blood cell count, although the initial antivenom treatment improved his coagulopathic parameters. Elevated white cell count was also observed in earlier reports of SBE (Lee and Sung, 2019; Yhi et al., 2013), and is a common observation for Russell's viper bite. In all the splenic rupture cases including the present study, hypotension was observed, and this may ultimately relate to the level of haemorrhage occurred in spleen. The development of abdominal pain and distension with or without tenderness along with features of shock are the warning signs of haemorrhage in visceral organs with or without rupture and haemoperitoneum.

Russell's viper (specifically from Southern India) venom largely contains phospholipase A_2 (PLA_2), snaclecs, serine proteases and metalloproteases as well as other minor components (Kalita et al., 2018). Russell's viper bites are well known to induce bleeding complications from the bite site and externally/internally from other organs throughout their geographical distribution (Jayanthi and Veerabasappa Gowda, 1988; Mukherjee et al., 2000). Most of the major components of Russell's viper venom affect blood coagulation by targeting various clotting factors and circulating platelets. The metalloproteases will affect the blood capillaries by digesting collagen and PLA_2 aggravates vascular complications (Frangieh et al., 2021; Gutiérrez et al., 2016). Therefore, the haemorrhage in spleen would have occurred as a collective action of multiple venom components. Since the spleen is acting as a natural blood filter and has inherent vascular nature, the haemorrhagic venom components might have caused excessive damage in spleen. Additionally, the non-enzymatic venom components might have enhanced the vascular permeability in the splenic capsule for venom components (Frangieh et al., 2021). The delayed result of 20-WBCT20 indicates abnormal blood clotting (Wedasingha et al., 2020). Moreover, recurrence of bleeding after treatment with antivenom is not uncommon as reported in similar cases (Kim et al., 2021; Lee and Sung, 2019; Yhi et al., 2013). Diverse enzymatic and non-enzymatic components might have collectively induced hypotensive effects secondary to internal bleeding and haemorrhagic shock (Frangieh et al., 2021). Hypotension might have also been compounded by the vasodilation effects of PLA_2 (Frangieh et al., 2021; Kakumanu et al., 2019). The patient's initial antivenom treatment immediately after the bite had improved his clotting parameters but prolonged prothrombin time and activated partial thromboplastin time two days after treatment potentially indicating that the coagulopathic effect of venom toxins has continued even after administering antivenom. Serine proteases present abundantly among viper venoms directly induce coagulopathy (Vaiyapuri et al., 2012). Although its method of action is proteolytic cleavage of specific blood components during coagulation (Matsui et al., 2000), further investigation is required to explore the reasons behind their coagulopathic parameters improving initially upon treatment followed by continuous deterioration despite subsequent to antivenom treatment and ultimately leading to splenic rupture. The patient's clinical improvement of coagulation and other parameters after treatment with antivenom strengthens the possibility that this effect indeed is due to SBE. Notably, we cannot rule out the possibilities of adverse effects that might have resulted from antivenom administration.

Although there was no bleeding or haematoma observed outside of spleen in this case, we cannot entirely rule out the possibility of bleeding from any other sites such as blood vessels in the abdominal cavity (Lucey et al., 2007). Moreover, the splenic rupture due to congestion and haemoperitoneum could have occurred independently without any direct impacts from SBE-induced complications. As detailed above, atraumatic, or spontaneous splenic rupture is a clinical diagnosis that is not uncommon in clinical settings, however, when it occurs, it is a life-threatening emergency that should be tackled promptly to save the patient (Lucey et al., 2007). Based on the emergency scenarios, a simple, easily available ultrasound scan could be used to ascertain haemoperitoneum and proceed with surgical procedures without any delay. In this case, the patient might have also had underlying health conditions such as an infection, microtrauma, perisplenic adhesions or tumour within the spleen (Husni and Turell, 1961) without any symptoms or previous diagnosis. The impact of these as well as other unnoticed health conditions may result in splenic congestion and subsequent rupture leading to excessive bleeding and haematoma following SBE. Here, SBE-induced complications may play indirect roles in induing splenic congestion and subsequent rupture. Further research to underpin the molecular mechanisms through which such splenic rupture and associated haemoperitenium occur in SBE victims will be highly beneficial to better understand the venom-induced pathophysiology on spleen and haemoperitoneum.

Together, this case reports a clinically rare event because the patient has initially responded to antivenom treatment, however, further complications worsened his normal health profile and resulted in splenectomy. Further investigation is required to determine the flow of venom components in and out of spleen to understand how these components escaped from antivenom neutralisation and how they could have caused excessive haemorrhage in spleen. The spleen and its membranes act as a blood filter, so it may be possible that large venom components were trapped in the vascular bed on their own or in complex with antivenom or target cell types. Lack of documentation of atraumatic splenic rupture in earlier series of viper (specifically, Russell's viper) envenomation could be due to the inter- and intra-species venom variations and the susceptibility of the individuals who might have pre-existing (known/unknown) health conditions. The recent reports of unusual clinical events following Russell's viper bites in India suggest that the variations in their venom components might be significantly higher than previously anticipated. These factors should also be considered in antivenom development to neutralise varying venom components from specimens living in different geographical locations. Moreover, the excessive bleeding and splenic rupture together with haemoperitoneum could have occurred due to internal bleeding and other complications in the abdominal cavity independently from venom toxins and SBE-induced coagulopathy. Updated training for medical students, clinicians and allied healthcare professionals by including case reports such as this will improve clinical diagnosis and management of such unusual complications of SBE and ensure patient safety and quality of care (Hughes, 2008). This will enable the healthcare professionals to provide timely interventions and thereby reduce the SBE-induced mortalities and morbidities.

Ethical statement: The data collection, consent form, and information sheets were approved by the Institutional Ethics Committee at Toxiven Biotech, Tamil Nadu, India (Reference number: ICMR-Toxiven Ethics 2021/1). A written consent was obtained from the patient to collect and publish the data presented in this article.

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Table 1: **Laboratory examination results for the patient at the time of admission in our emergency department**

Investigation	Results	Unit	Normal range
Haemoglobin	6.0	gms%	13.0 – 16.0
Total RBC count	5.49	Millions/µL	4.00 – 5.00
HCT	42.9	%	41.00 – 50.00
MCV	78.1	fl	81.10 – 96.00
MCH	26.2	pg	27.20 – 33.20
MCHC	33.6	%	32 - 36
Total WBC count	16.40	$x10^3$ Cells/µL	4.00 – 11.00
Neutrophils	3.98	$x10^3$ Cells/µL	2.0 to 7.0
Lymphocytes	1.71	$x10^3$ Cells/µL	1.0 to 3.0
Monocytes	0.52	$x10^3$ Cells/µL	0.1 to 0.8
Eosinophils	0.42	$x10^3$ Cells/µL	0.02 to 0.5
Basophils	0.05	$x10^3$ Cells/µL	0.02 to 0.1
Neutrophils	59.6	%	55 – 75
Lymphocytes	25.6	%	15 – 30
Eosinophils	6.3	%	1 - 5
Monocytes	7.8	%	2 - 10
Basophils	0.7	%	Up to 1
Platelet Count	189	$x10^3$ Cells/µL	150 - 450
MPV	8.4	fl	6.5 - 12.0
PDW	8.8	fl	9.0 - 13.0
Urea	23.54	mg/dL	15 - 40
Creatinine	0.71	mg/dL	0.7 - 1.4
Uric Acid	6.9	mg/dL	3.4 - 7.2

RBC, red blood cell; 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.

316 **Figure 1**
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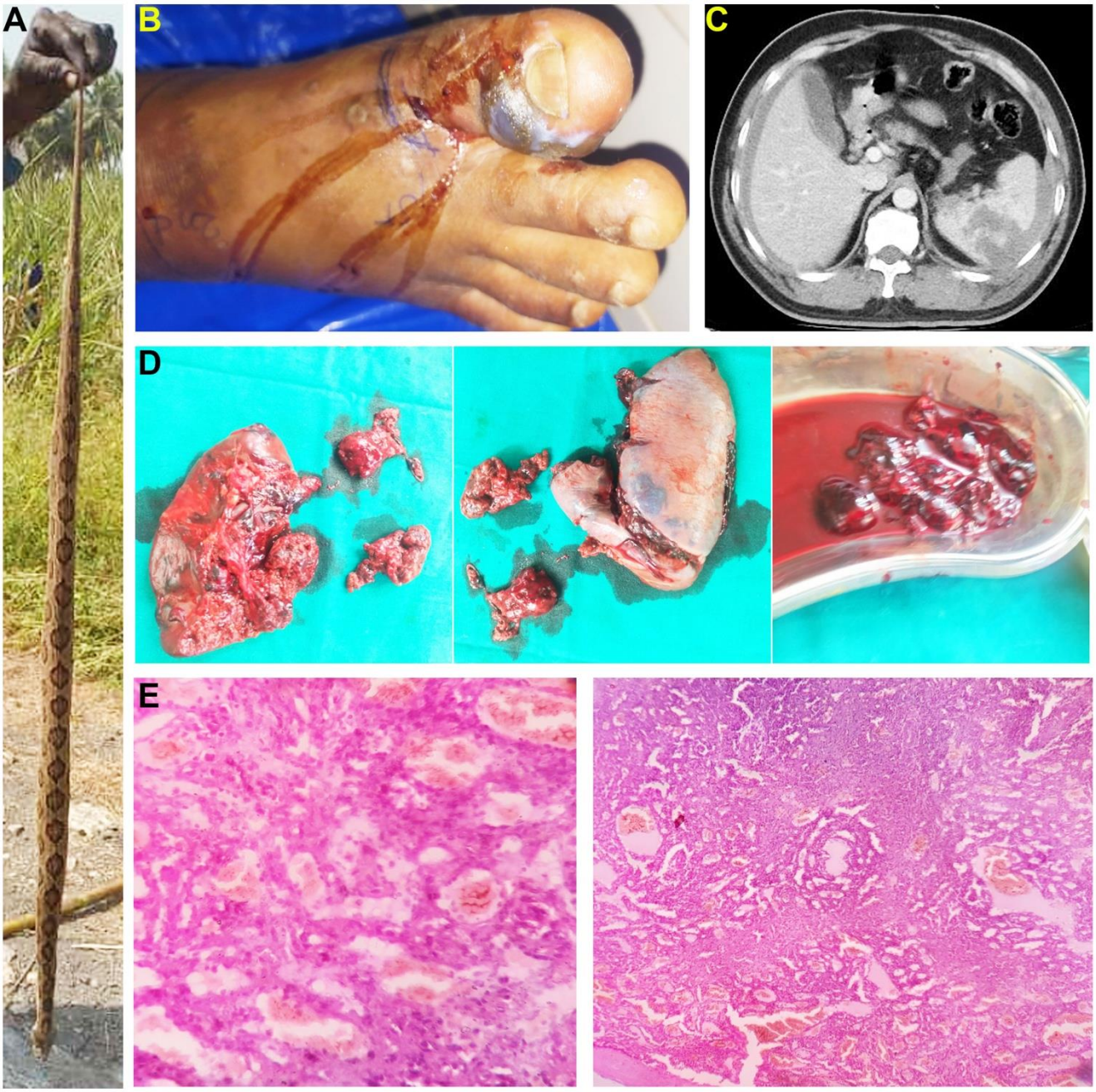

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320 **Figure 1**: **A**, the offending snake was identified as a Russell's viper by a herpetologist. **B**, local swelling
321 along with bluish focal haemorrhagic bulla was observed at the bite site of victim. **C**, the CT examination
322 highlights fluid collection in Morison's pouch, splenorenal access and suprapubic space. **D**, excessive
323 haemorrhage observed at the surface of spleen following splenectomy. **E**, haematoxylin and eosin stain
324 of sections of removed spleen confirms the congestion in red pulp and hilum due to excessive
325 haemorrhage without any infarcts.