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Splenic rupture and subsequent splenectomy in a young healthy victim following Russell's viper bite

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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 A₂ 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

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

65 Case presentation

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

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

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

115 **Case discussion**

116 There are multiple causes for atraumatic or spontaneous splenic rupture such as microbial
117 infection, tumour growth, hyperplasia of splenic cells, physical activities such as weightlifting and rarely,
118 some physiological processes including pregnancy (Halliday et al., 2020; Kaniappan et al., 2018; Lam
119 et al., 2014; Lieberman and Levitt, 1989; Renzulli et al., 2009; Rueda-Esteban et al., 2020). However,
120 the adverse effects of SBE on spleen are rarely encountered. Prior to this study, a cobra bite-induced
121 splenic congestion was observed in a victim in 19th century (as reviewed in (Feola et al., 2020)).
122 Similarly, a total of four cases of splenic rupture following bites from unidentified snake species one to
123 five days after receiving antivenom were reported from South Korea (Kang et al., 2014; Kim et al., 2021;
124 Lee and Sung, 2019; Yhi et al., 2013). Among these cases, three underwent splenectomy (Kang et al.,
125 2014; Kim et al., 2021; Yhi et al., 2013) and in one case, it was averted by successful angioembolisation
126 of splenic artery (Lee and Sung, 2019). To our knowledge, splenic rupture and subsequent splenectomy
127 in Russell's viper bite victims has not been previously reported. Moreover, splenic rupture in a SBE
128 victim due to excessive haemorrhage following antivenom treatment is an unusual clinical event. Hence,
129 we report this case to highlight this rare complication following a Russell's viper bite in India.

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

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

158 Russell's viper (specifically from Southern India) venom largely contains phospholipase A₂ (PLA₂),
159 snakecs, serine proteases and metalloproteases as well as other minor components (Kalita et al.,
160 2018). Russell's viper bites are well known to induce bleeding complications from the bite site and
161 externally/internally from other organs throughout their geographical distribution (Jayanthi and
162 Veerabasappa Gowda, 1988; Mukherjee et al., 2000). Most of the major components of Russell's viper
163 venom affect blood coagulation by targeting various clotting factors and circulating platelets. The
164 metalloproteases will affect the blood capillaries by digesting collagen and PLA₂ aggravates vascular
165 complications (Frangieh et al., 2021; Gutiérrez et al., 2016). Therefore, the haemorrhage in spleen
166 would have occurred as a collective action of multiple venom components. Since the spleen is acting
167 as a natural blood filter and has inherent vascular nature, the haemorrhagic venom components might
168 have caused excessive damage in spleen. Additionally, the non-enzymatic venom components might
169 have enhanced the vascular permeability in the splenic capsule for venom components (Frangieh et al.,
170 2021). The delayed result of 20-WBCT20 indicates abnormal blood clotting (Wedasingha et al., 2020).
171 Moreover, recurrence of bleeding after treatment with antivenom is not uncommon as reported in similar
172 cases (Kim et al., 2021; Lee and Sung, 2019; Yhi et al., 2013). Diverse enzymatic and non-enzymatic
173 components might have collectively induced hypotensive effects secondary to internal bleeding and
174 haemorrhagic shock (Frangieh et al., 2021). Hypotension might have also been compounded by the
175 vasodilation effects of PLA₂ (Frangieh et al., 2021; Kakumanu et al., 2019). The patient's initial
176 antivenom treatment immediately after the bite had improved his clotting parameters but prolonged
177 prothrombin time and activated partial thromboplastin time two days after treatment potentially indicating
178 that the coagulopathic effect of venom toxins has continued even after administering antivenom. Serine
179 proteases present abundantly among viper venoms directly induce coagulopathy (Vaiyapuri et al.,
180 2012). Although its method of action is proteolytic cleavage of specific blood components during
181 coagulation (Matsui et al., 2000), further investigation is required to explore the reasons behind their
182 coagulopathic parameters improving initially upon treatment followed by continuous deterioration
183 despite subsequent to antivenom treatment and ultimately leading to splenic rupture. The patient's
184 clinical improvement of coagulation and other parameters after treatment with antivenom strengthens
185 the possibility that this effect indeed is due to SBE. Notably, we cannot rule out the possibilities of
186 adverse effects that might have resulted from antivenom administration.

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

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

223 **Ethical statement**: The data collection, consent form, and information sheets were approved by the
224 Institutional Ethics Committee at Toxiven Biotech, Tamil Nadu, India (Reference number: ICMR-Toxiven
225 Ethics 2021/1). A written consent was obtained from the patient to collect and publish the data presented
226 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/ μ L	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	$\times 10^3$ Cells/ μ L	4.00 – 11.00
Neutrophils	3.98	$\times 10^3$ Cells/ μ L	2.0 to 7.0
Lymphocytes	1.71	$\times 10^3$ Cells/ μ L	1.0 to 3.0
Monocytes	0.52	$\times 10^3$ Cells/ μ L	0.1 to 0.8
Eosinophils	0.42	$\times 10^3$ Cells/ μ L	0.02 to 0.5
Basophils	0.05	$\times 10^3$ Cells/ μ L	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	$\times 10^3$ Cells/ μ L	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

311
 312 RBC, red blood cell; HCT, haematocrit; MCV, mean corpuscular volume; MCH, mean corpuscular
 313 haemoglobin; MCHC, mean corpuscular haemoglobin concentration; WBC, white blood cells; MPV,
 314 mean platelet volume; PDW, platelet distribution width.
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Figure 1



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