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CASE REPORTS

UNUSUAL CAUSE OF CAPILLARY LEAK SYNDROME WITH SHOCK IN A CHILD

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ABSTRACT

A 12 years male child with lethargy, vomiting, and pain abdomen presented with compensated shock at our emergency department. Shock managed with 2 boluses of normal saline and started on inotropes. Ejection fraction was 35%. Child was intubated and shifted to pediatric intensive care unit. Initial diagnosis was septic shock/dengue shock syndrome/Multisystem inflammatory syndrome associated with Covid 19 (MIS- C). Child showed chemosis, parotitis, edema of eyelids, hemoconcentration, bilateral pleural fluid, ascites, hypoalbuminemia, and progressed to multiorgan failure. On examination, two fang marks were noted near the gluteal fold. Capillary leak syndrome with shock following snake envenomation was thought of Child received 30 vials of ASV with supportive management. Developed acute kidney injury and coagulopathy and was started on hemodialysis. Child received IV immunoglobulin. Despite the efforts, child died on the fourth day of hospitalization.

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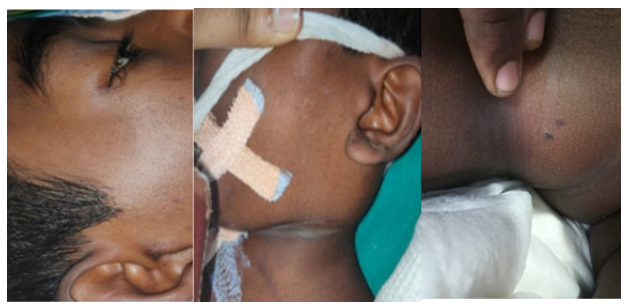
Renal failure, Capillary leak, snake envenomation, shock

Case Report

Shock is a common medical emergency in children. Capillary leak syndrome with shock is a common presentation in Dengue shock syndrome or sepsis. Coagulopathy, cellulites and renal failure are well known complications of snake envenomation.[1] Capillary leak syndrome with shock is a fatal complication that has been less frequently reported and remains as an under recognized complication. One of the common causes of death in viper bites is due to capillary leak syndrome. The following case report is snake envenomation presenting with shock and capillary leak syndrome.

A 12-year-old boy presented to the emergency department with vomiting, pain abdomen, and lethargy. He was treated elsewhere as acute abdomen and referred on day 6 of illness. He was afebrile, verbal responsive, heart rate was 94/ minute, respiratory rate was 28/minute, blood pressure was 100/80 mmHg. His peripheral pulses were feeble and was cool below knee. There were no petechiae, purpura or ecchymosis in skin. In view of gastrointestinal symptoms with lethargy and shock, initial differential diagnosis included dengue shock/ septic shock / multisystem inflammatory syndrome associated with COVID 19. Absence of fever, eschar, hepatomegaly, and lymphadenopathy was not supportive of an acute febrile illness with tropical infection. Shock was corrected with boluses up to 20 ml/kg normal saline and started ECG monitor showed wide complex tachycardia suggestive of ventricular tachycardia and child reverted to normal rhythm with lidocaine. Child was intubated and shifted to pediatric intensive care unit with inotropes. Child showed chemosis, parotitis, edema of eyelids. (Figure 1) . There was no significant past medical illness or allergy. Laboratory evaluation revealed hemoconcentration, bilateral pleural fluid, ascites, hypoalbuminemia, and elevated urea creatinine. Electrolytes and calcium were normal. Bedside screening echo revealed poor left ventricular function with an ejection fraction of 35%. On head to toe examination two healing fang marks were noted in the gluteal region near the anal orifice. Retrospective history revealed that child used to squat in open field to defecate and the child had passed stools 6 days back. Mother was unaware of any history of bite. Child had chemosis, bilateral parotid swelling, periorbital puffiness, and was anuric within 12 hours of hospital stay.

Figure 1. Periorbital edema and parotid swelling and the fang mark.



Management

Initial hematocrit was 23.6 and subsequently 35.5, 40.2, and 43.2. Repeat echo showed an ejection fraction of 30% with normal coronary Z scores. There was no pericardial effusion. Child continued to be in shock and inotropes were escalated to adrenaline and nor

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Table 1. Showing the laboratory parameters.

Parameter	Trend of values											
Total count cellsx109/L	7100	12000	17700	18400	17900	21120	14000	23200	21900	14900	18700	
Hb gm/L	7.6	11.8	13.2	14	12	10	8.8	8.7	8.1	7	7.5	
Hct	23.6	35.5	40.2	43.1	36.8	30.6	27	26.7	25.7	23.2	24.1	
Platelet cells x109/L	183	243	182	162	136	24	48	34	21	18	19	
ESRmm/hr	1 mm					1 mm				2 mm		
Urea mmol/L	40	63	90	103	130	133	144	160	195	211	237	
Creatinine µmol/L	0.3	0.9	2.1	3	4.2	5	5.2	5.7	6.4	6.5	7.2	
Sodium mmol/L	143	130	129	127	130	131	128	134	137	149	129	
Potassium mmol/L	3.4	5.3	5.4	5.6	5.3	5.1	4.8	5.0	5.4	6.2	6.6	5.4
Calcium mmol/L	4.3		8.3		9		7		8.2		9.3	
SGOT U/L	83	159	6088	14449	22278		7989	4095	2526	895	447	
SGPT U/L	35	76	3642	6807	9382		6363	4882	3691	2159	1475	
Serum Bilirubin µmol/L	0.6		0.7	0.9			2.6	2.7	2.9	3.9	3.9	4.5
Serum Protein g/L			5.4				5.1					
Serum Albumin g/L			3.2				2.8					
Ferritin pmol/L	385						4292					
LDH U/L					456		987					
Blood culture			No growth									
Urine culture			No growth									

adrenaline. In view of elevated renal parameters at 24 hours of admission child was started on hemodialysis. The laboratory parameters are summarized in Table 1. Child had elevated liver enzymes, hyperferritinemia, hypofibrinogenemia, and hypoalbuminemia. CRP was negative. Chest X-ray showed cardiomegaly. Dengue serology, MAT for leptospirosis, scrub typhus serology, widal test, blood, and urine culture reports were negative. Viral panel for myocarditis including mumps serology was negative. Covid RT PCR a was negative and covid antibody was non-reactive. Urine protein creatinine ratio was.[3,4]

Discussion

Though increasing hematocrit, fluid accumulation, and elevated inflammatory markers were suggestive of dengue or septic shock, lower Hb at admission without evidence of bleeds, absence of fever despite elevating ferritin, higher leucocyte counts, dengue NS1 and IgM negative results were pointing against dengue in this child. The fluid accumulation had an unusual pattern involving periorbital region with bilateral parotid swelling. Despite significant fluid overload due to myocardial dysfunction and renal failure, hematocrit was in increasing trend. Hypoalbuminemia was also present. Clinical and lab findings were suggestive of capillary leak syndrome. The child had worsening AKI and coagulopathy since admission along with a healing fang mark , envenomation was thought of.[2] Though very rare, envenomation with viper is known to cause capillary leak syndrome. Child received 30 vials of anti-snake venom (ASV). But it was already day 6 of illness by the time he reached our institute. As the child had no response to ASV intravenous immunoglobulin was also given. Despite all above efforts, child developed multiorgan dysfunction and died.

Capillary leak syndrome is also called as Clarkson's syndrome is the major cause of mortality in patients with viper envenomation. The protein component of

venom causes systemic vascular endothelial damage which results in plasma leak and edema. There are two vascular apoptosis–inducing proteins [VAP1 and VAP2] of which vascular apoptosis-inducing protein 2 [VAP2] is more specific to endothelial cells. Another postulated mechanism is the cytokine activation by the venom.[3,4]

The criteria for diagnosis include the presence of bilateral parotid swelling, chemosis and periorbital edema following snake bite with three or more of the following: systolic blood pressure <90 or fall in MAP by 20mmHg, hematocrit >45% or >20% elevation from baseline, spot PCR >1, serum albumin <3 g/dl, third space fluid collection.[3] The clinical features of capillary leak start appearing from 2nd or 3rd day. The typical fluid accumulation over face in the form of parotid swelling, chemosis, and periorbital edema has been termed as "viper head appearance".

The mortality rate is reported as high as 50 to 60%[4,5] It has been postulated that a vascular apoptosis-inducing component of Russell's viper venom that is not neutralized by the commercially available ASV is responsible for its high fatality rate. Other modalities of management for capillary leak syndrome in literature includes intravenous immunoglobulin, plasmapheresis, and methylprednisolone. This complication is frequently recognized in states like Kerala, whereas in Tamil Nādu there are no case reports of capillary leak syndrome so far and hence remains an under recognized complication. In any child with features of shock, facial and parotid edema, hemoconcentration, fluid leak with acute kidney injury in the absence of fever one should think of this rare complication.

Lessons learnt

- Haemoconcentration and fluid leak with shock can be a rare complication with snake envenomation
- Beyond infectious causes envenomation should be a differential diagnosis in unexplained renal failure and coagulopathy.
- Capillary leak syndrome following snake envenomation can mimic dengue shock

Compliance with Ethical Standards

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Conflict of Interest None

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