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A Rare Case of Hemolytic Uremic Syndrome in Patient with Russel Viper Snake Bite

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Running Title: HemolyticUremic Syndrome

Abstract: ***Snake bite is predominantly an occupational hazard and causes severe health issues. Snake poisoning in India is a significant and prevalent cause of Acute Kidney Injury (AKI). Mechanisms such as haemodynamic disturbances, direct tubular toxicity, coagulopathy, haemoglobinuria, and myoglobinuria can cause AKI after bites by snakes. Renal pathologic findings include acute tubular necrosis, cortical necrosis, interstitial nephritis, glomerulonephritis, and vasculitis. Thrombotic Microangiopathy (TMA) as a cause of snakebite-induced AKI is very rare. Haemolytic Uremic Syndrome (HUS) is a clinical disease that includes TMA, thrombocytopenia, and AKI as a triad. The HUS is a heterogeneity of illnesses with different aetiology which results in presentation, therapy and outcomes variance. Hereby, authors report a case of a 60-year-old male Who was bitten by Russell viper and developed HUS. Patient eventually progressed to AKI and then managed at our setup successfully after 19 days of continuous monitoring and management by the supportive measures, plasmapheresis and hemodialysis. HUS should be taken into account as a probable cause of AKI following a snake bite.***

Keywords: Snake Bite, Hemolyticuremic Syndrome, Acute kidney Injury, Schistiocytes, Plasmapheresis

1. Case Presentation

History

A 60-year-old male hypertensive non diabetic, farmer by occupation came to hospital with alleged history of snake bite two hours back on left ring finger while working on field and complain of abdominal pain, black stool, dysuria, generalized weakness, nausea, vomiting. The patient's co-worker identified the snake as Russell's viper, but the snake was not captured. He then Visited to private hospital and the patient was treated by 25 polyvalent ASV injection which neutralises four most important venomous species found in India in one hour after reconstitution of 10 vials of lyophilised ASV in 250 mL of isotonic saline intravenously administrated and was repeated till 25 vials. No adverse effects were detected with its administration then he came to LG Hospital due to economic issues.

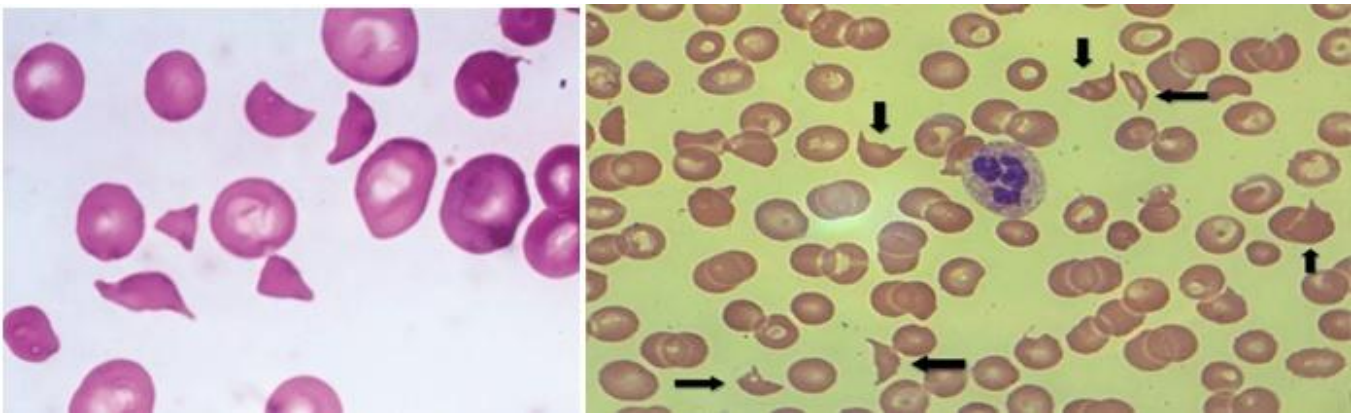
Examination

On examination, two fang marks were present over the left ring finger, patient had swelling over local site along with erythema approximately 2x1x1.5cm, raised local temperature and marked tenderness. Pulse was 120/minute regular, Blood Pressure (BP)-182/100 mmHg in right arm. systemic examinations were normal. There was no neurodeficitnoted.

Investigations

On admission investigations revealed, Haemoglobin (Hb) 14.5 gm%, Total Leukocyte Count (TLC) 8300/mm3, platelet count 97000/mm3, serum creatinine 1.1 mg%, urea 38 mg%, sodium 137mEq/L, potassium 4.2 mEq/L. Coagulation profile and Liver Function Test (LFT) was within normal limits. There was no evidence of schistocytes on peripheral smear.

3 day later, patient developed anuria (urine output less than 100 mL/day) and facial puffiness. Repeat laboratory investigations revealed Hb 5.6 gm%, Platelet count 40000/mm3 and TLC 4300/mm3 ,serum creatinine 10.97 mg%, urea 312 mg%, sodium 140 mEq/L and potassium 5.8 mEq/L. Peripheral smear was suggestive of schistocytes (arrow headed)[1] Seum Lactatate Dehydrogenase- 1530 IU, Blood culture results were negative for E.coli. direct and indirect Coomb's test was negative.



Schistiocytes seen in peripheral smear of Hemolytic Uremic Syndrome

Management

In view of rapidly declining renal function and in view of hyperkalemia the patient was taken for haemodialysis. Investigation findings together with hypertension, thrombocytopenia, microangiopathic haemolytic anaemia,Acute kidney injury prompted the diagnosis of HUS. A Disintegrin and Metalloproteinase with Thrombospondin Motifs type 1, member 13 (ADAMTS13) level and genetic studies were not done, owing to the unavailability of resources. Patient was continued on hemodialysis and plasmapheresis. alternatively, 4 cycles of Hemodialysis and 3 cycles of plasmapheresis done and patient gradually recovered, with an increase in platelet count, haemoglobin. continuous monitoring of vitals and the reports were done and He was discharged with the normal creatinine and the hemoglobin level after 19 days.

2. Discussion of HUS

Hemolyticuremic syndrome (hus) is a triad consisting of microangiopathichemolyticanemia, thrombocytopenia, and acute renal failure.HUS typically occurs in children following a diarrhoeal episode which is most commonly a hemorrhagic type. Survival greatly improved with the advent and improvement of dialysis and kidney transplantation. However, HUS remains a leading cause of acute renal failure in children and is increasingly recognized as a cause of renal failure in adults.

Typical HUS

In most cases of HUS, the cause is activity of toxigenic proteins that have deleterious effects on endothelial cells, particularly those of colon and kidney. toxins of Shigella, shigella dysenteriae 1 toxin and EHEC O157:H7 are identified to cause typical HUS.

Atypical HUS (aHUS)

In secondary or atypical HUS(aHUS), any coexisting infections or a disease is associated with manifestation of HUS . Pathogenesis of secondary HUS has not been intensively studied, but complement is involved in some cases.aHUS is the severe disease with upto 15% of mortality in acute phase and upto 50% of cases progressive to end stage renal disease.

The most frequently Associated diseases that lead to clinically evident secondary HUS include infections, especially those caused by Streptococcus pneumoniae, and the influenza virus. These infections are considered as causes and triggers for aHUS.

In addition to infections, secondary HUS may be associated with other toxins like russelviper venom, transplantation (solid organ or bone marrow), autoimmune disease, cancer, pregnancy, and the use of certain cytotoxic drugs. The common feature for these coexisting diseases or conditions is that they may cause direct cell damage, promote activation of the complement system in general, or enhance activation of complement on self cells.

There is also a genetic basis of Atypical HUS as evident from gene study of family members who developed HUS which showed mutation in the several genes that encode complement regulatory proteins: - complement factor H(CFH), complement factor I(CFI),complement factor B(CFB) , membrane cofactor protein(MCP), complement component C3, thrombomodulin and others. When HUS is due to inheritted abnormality relapses are possible and in such cases prognosis is always serious.

During episode of infection or some other trigger the deficiency of one of the complement regulators becomes evident

Differential Diagnosis

1) TTP
2) DIC
3) HELLP Syndrome

Snake envenomation & AKI

Snake envenomation is a major and common cause of AKI. There are more than three million snake bites per year, and more than 1,50,000 deaths worldwide[2].In rural India, snake envenomation is an occupational hazard, with most bites occurring in farms fields during the night. There are four families of poisonous snakes: Elapidae, Viperidae, Hydrophidae and Colubridae. The most common clinical effects of elapid venom are neurotoxic, whereas those of vipers are vasculotoxic and Hydrophiinae or sea snakes are myotoxic. Viper bites are the most common in India and the incidence of AKI following Russell's viper bites is 13-32% [3]

Pathogenesis of AKI in snake envenomation includes hypotension, haemolysis, rhabdomyolysis, DIC, direct

cytotoxic effect of the venom, sepsis, haemodynamic alterations, and cell damage triggered by the release of proinflammatory cytokines and vasoactive mediators.Plasma exchange can be considered in patients who are refractory to traditional ASV treatment.

3. General Management

Management of HUS is supportive and chiefly involves dialysis for individuals with renal failure. Acute medical issues involve the management of renal failure and hypertension, the maintenance of fluid status in the face of renal failure, and the treatment of fever and catabolic status. Adult HUS and its underlying illnesses respond poorly to various forms of medical therapy.

Eculizumab (Soliris) is the first treatment approved by the US Food and Drug Administration for adults and children with atypical hemolyticuremic syndrome (aHUS). Approval was based on data from adults and children who were resistant or intolerant to, or receiving, long-term plasma exchange/infusion.

4. Conclusion

Generally HUS most commonly occurs after e.coli infections but in our case snake bite induced hemolyticuremic syndrome which is rare, which was managed by supportive therapy, dialysis and plasmas exchange.

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