



Case Report

# Secondary capillary leak syndrome - Plasmapheresis: Is it the answer?

Mary Grace<sup>1\*</sup>, K J Jacob<sup>2</sup>, Jayakumar<sup>3</sup>, Arun P<sup>4</sup>

<sup>1</sup>Associate Professor, <sup>2</sup>Additional Professor, <sup>3</sup>Associate Professor, <sup>4</sup>Junior Resident  
Department of Medicine, Government Medical College, Thrissur, India

\*Corresponding author email: [nc.grace@yahoo.in](mailto:nc.grace@yahoo.in)

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## Abstract

Capillary leak syndrome is a dreaded complication of snake bite. Various modalities of treatment have been tried, but prognosis is very poor. We have reported here a case of poisonous snake bite complicated with capillary leak, which survived following plasmapheresis.

## Key words

Snake bite, Capillary leak, Plasmapheresis.

## Introduction

Capillary leak syndrome (CLS) also known as Clarkson's syndrome, was first described by Dr. Bayard Clarkson in 1960. It is characterized by hypoalbuminemia without albuminuria, hemoconcentration, hypotension and generalized edema. Capillary leak syndrome is a dreaded complication of snake bite. Various modalities of treatment have been tried, but prognosis is very poor.

## Case report

28 years old male admitted with snake bite on medial aspect of left leg near medial malleolus. At the time of admission the patient had abdominal pain and vomiting with pain at the site of bite. On examination, there was local

edema. There was no evidence of neurotoxicity or bleeding manifestations or decrease in urine output. In view of the evidence of local and systemic envenomation, he was given 20 vials of antsnake venom. He subsequently went into oligo anuric state with serum creatinine value of 1.4 mg%, for which renal replacement therapy in the form of hemodialysis was initiated. On the second day he developed chemosis, parotid swelling, right sided pleural effusion along with severe leukocytosis (total count 39000/cumm) and hemoconcentration (Hemoglobin 15 g%, PCV 46%). On the third day, he developed bleeding manifestations in the form of hematuria, malena, subconjunctival hemorrhage. Laboratory investigations revealed thrombocytopenia (platelet count 30,000/cumm). The coagulation parameters



were persistently normal. On the fourth day, patient complained of tightness in the abdominal wall and both forearms. CPK value was 2400 units/litre. Myoglobinuria was not evident. In view of capillary leak syndrome secondary to ophitoxemia, we started him on theophylline, leukotriene antagonists, and high dose pulse steroid therapy. Despite hemodialysis and disease modifying treatment for capillary leak, renal function tests and platelet counts showed a worsening trend. Hb dropped from 15 g% to 8.9 g% and then 7.6 g%. Peripheral smear did not show any evidence of hemolysis, LDH level was normal (54 units/liter). Patient went into hypotension, with worsening chemosis, hemoconcentration, hypoalbuminemia (serum albumin 2.8 g%, with no evidence of albuminuria) and rising serum creatinine. Daily plasmapheresis and replacement with fresh frozen plasma was done along with continuing SLED (slow low efficient dialysis). After two days of plasmapheresis patient started improving. Within one week of daily plasmapheresis with renal replacement therapy, all the laboratory parameters returned to normal and the patient could be discharged.

## Discussion

Capillary leak syndrome (CLS) also known as Clarkson's syndrome, was first described by Dr Bayard Clarkson in 1960. It is characterized by hypoalbuminemia without albuminuria, hemoconcentration, hypotension and generalized edema. The underlying mechanism is capillary hyper permeability and extravasation of plasma, containing macromolecules up to 900 kD. CLS can be primary (idiopathic) or secondary to conditions like poisonous snake bite (ophitoxemia) [1, 2] and dengue hemorrhagic fever. Toxins and enzymes present in the snake venom cause increase in the capillary permeability leading to leakage of plasma from the intravascular space. There are three phases in the clinical presentation of capillary leak

syndrome. First is the prodromal phase when the patient manifests irritability, fatigue, myalgia, abdominal pain, thirst and syncope. Second is the phase of capillary leakage with marked extravasation of intravascular fluid. Marked hypotension, generalized edema, pleural and pericardial effusion, ascites, compartment syndrome, rhabdomyolysis, renal failure can occur in this phase. Renal failure is a common complication. The mechanisms of renal failure include hypotension induced reduced glomerular filtration, acute tubular necrosis and rhabdomyolysis associated pigmenturia. This stage usually lasts for 1-4 days. Third phase is characterized by return of fluid back into the intravascular space and associated polyuria and pulmonary edema. This patient had features of envenomation followed by features like generalized edema, pleural effusion, compartment syndrome, rhabdomyolysis and renal failure [3]. The possibility of thrombotic thrombocytopenic purpura was considered, but there was no evidence of hemolysis. The persistently normal coagulation profile ruled out disseminated intravascular coagulation. Treatment of CLS includes anti-snake venom and supportive care. Disease modifying agents being used in treatment of capillary leak are theophylline, leukotriene antagonists, terbutaline, plasmapheresis, prostacycline, corticosteroids and intravenous immunoglobulin (IG). There is no proven therapy for CLS. The prognosis remains very poor. We postulated that early recognition of CLS and prompt institution of plasmapheresis and use of fresh frozen plasma might have contributed to the successful outcome in this patient.

## Conclusion

Capillary leak syndrome is a grave complication of snake bite with no proven treatment modalities. Early recognition and institution of plasmapheresis along with replacement with



fresh frozen plasma might be life saving in this fatal situation.

### References

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