

CASE REPORT**DELAYED ONSET RENAL FAILURE FOLLOWING
MULTIPLE BEE STINGS****Pratap Patra¹; Uma Nayak²**¹Assistant Professor; ²Additional Professor

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ABSTRACT

Bee sting is an uncommon pediatric emergency encountered worldwide and capable of causing acute renal failure, multiorgan dysfunction and occasionally death. We had a two year male child who suffered from multiple bee stings and delayed onset acute renal failure, survived following peritoneal dialysis.

Key Words: Bee Stings;Envenomation;Delayed Onset Renal Failure

INTRODUCTION

The dangers of Hymenoptera (wasp, bees, hornets, and yellow jackets) have long been known as a potential environmental hazard.^[1]The sting usually follows mild self-limiting local reaction however; in individual who are stung for multiple times and when there is massive bee envenomation it leads to vomiting, diarrhea, dyspnoea, hypotension, circulatory failure and death. Acute renal failure which developed following bee sting may not manifest clinically as well as biochemically during early period of Hospitalization. We report a case of acute renal failure following multiple bee stings which was delayed onset and only a single similar case have been reported till date.^[2]

CASE REPORT

A two year male child was stung by swarm of bees while playing along with sister on field when a large beehive was hit by a bird on a nearby tree. Following which he developed swelling over the face, inability to open eyes, 4 to 5 episode of vomiting with high grade fever and altered sensorium. On admission to emergency department his pulse rate was 124/mt, respiratory rate 58/min and Blood pressure

was 120/80mm Hg. He also had scalp edema, swelling over the face with few leftover stings which were removed immediately. There was sticking of eye lid with thick discharge. He was stuporous and modified Glasgow coma scale score was 5/15 with intermittent tonic posturing. Central nervous system examination did not reveal any other abnormality. Further Ophthalmologic evaluation revealed eyelid edema, matted eye lashes with discharge, conjunctiva congestion and hazy cornea. All other systemic examination was within normal limit. Initial investigation revealed Hb 7gm% and there was microcytic hypochromic anemia.TLC was 10,000 cumm, DLC 72/ 20 /8/00, Platelet count 1.8 lacs, serum sodium 134/meq/l, and potassium 4.7 meq/l,blood urea 54mg/dl, serum creatinine 0.6 mg/dl. Routine and microscopic examination of urine, chest x-ray and computerized tomographic scan of head was within normal limit. He was initially treated with intravenous corticosteroid, antihistaminic and antibiotics. His sensorium improved by day 4 of admission and tachypnea settled down. Ophthalmic condition was managed by antibiotic eye drop. Seven days later the child developed puffiness of both eyes along with severe oliguria for which intravenous fluid bolus followed by lasix challenge was given however there was no improvement in urine output. Repeat renal

function test revealed blood urea of 290mg/dl, creatinine 3.9mg/dl, Na⁺ 141 meq/l, K⁺ 7.1meq/l, C₃ level; routine and microscopic examination of urine was within normal limit. Hyperkalemia was managed by intravenous calcium gluconate, Asthaline nebulisation and subsequently peritoneal dialysis was started. After 50 cycle of peritoneal dialysis his electrolytes became normal, urine output improved remarkably but urea and creatinine continued to be elevated till day 20 of admission. His renal function became absolutely normal on day 23 of admission and subsequently he was discharged from the hospital. On follow up after a week, repeat renal function test were within normal limit.

DISCUSSION

The reaction following hymenoptera stings are grouped in to local, toxic, unusual and anaphylactic reactions. It has been estimated that systemic toxicity may occur with a minimum of 50 stings in adults however; the minimum number of stings causing systemic toxicity in children is less clearly defined. The early toxic reactions are nausea, vomiting, hemolysis, kidney failure and disseminated intravascular coagulation. Delayed toxic reaction refers to a patient who is asymptomatic after massive envenomation but later on develop the evidence of organ dysfunction.^[3]

Bee venom contains a complex of mixture of more than 20 identified enzymes, peptides and active amines. Mellittin and phospholipase which constitute up to 60% of its component has hemolytic, vasoactive, and contractile and cell membrane lytic effect. Mellittin act on the apical membrane cell transporters of proximal tubular cell of the kidney thus reduce the cell viability and inhibit the activity of renal transporter. Acute renal failure result either from direct toxic effect on proximal tubule of Mellittin or secondary to vasoconstrictive effect of it.^[4]

Acute Renal failure which develops following bee stings is biphasic in nature.^[5] Early onset renal failure occur secondary to rhabdomyolysis, hemolysis, hypotension or to anaphylactic shock mediated by the toxin. The mechanism of delayed onset renal failure is less clearly defined and it is thought to be secondary to non-immunological IgE mediated reaction with deposition of immune complex (Type 3 hypersensitive reaction) leading to nephritis.^[2] Though the index case had features of early toxicity in the form of altered sensorium, tachypnea and hypertension soon after the stings but renal failure was only manifested after a week of hospitalization and there was no supportive evidence for nephritis. It is possible that in our case the acute renal failure was the result of delayed toxic manifestation of Mellittin.

To conclude, there can be delayed onset acute renal failure in children following multiple bee stings as a result of direct toxic effect of the venom on the renal tubule, therefore require meticulous monitoring of renal function and follow up at least for a two week period.

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