

Nephrotic syndrome induced by bee sting

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Manifestations of bee sting are varied ranging from local pain and swelling to anaphylaxis. Renal involvement has also been well documented in the literature. Nephrotic Syndrome is known to be triggered by various immunological stimuli including bee sting. We are reporting an 8-year-old child who developed Nephrotic Syndrome 6 days following the bee sting. The diagnosis was established on the temporal relationship of the sting and onset of anasarca. High dose of oral Prednisolone was started in the dose of 2mg/kg/day on which the child showed prompt remission. The exact cause of Nephrotic Syndrome following exposure to bee venom is not clearly stated in the literature, though it is postulated that bee venom mediated immunological disturbances lead to increased permeability of glomerular basement membrane and heavy proteinuria.

Keywords: Nephrotic Syndrome, Bee sting

Case Report

An 8-year-old boy presented in Paediatric emergency with massive swelling over body and decreased urine output. The child was apparently well 10 days back when he had a bee sting while playing on his right forearm, following which he developed a swelling, pain and redness over local area. Symptomatic treatment was taken and the local symptoms gradually subsided. Six days later, child developed swelling over face, starting from periorbital area which rapidly progressed to the whole body over a period of 2 days. Child also had oliguria since 1 day. There was no history of allergy/ atopy and no significant past history.

On examination, the child had generalized bilaterally symmetrical pitting oedema along with ascites. The sting mark was present on the posterior aspect of right forearm with minimal oedema (Figure 1). The cardiovascular system was normal. There was no organomegaly. On investigation, there was heavy proteinuria (heat test for urinary protein estimation was 4+). There was no evidence of haematuria. Differential leucocyte count showed raised Eosinophil count of

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Figure 1: Showing Bee sting mark on the forearm with minimal swelling



14%. Serum cholesterol was 655 mg/dl, serum albumin was 0.89 gm/dl and serum creatinine was normal. ESR was 96 mm in 1st hour. On the basis of clinical

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assessment and laboratory reports, the child was diagnosed as Nephrotic syndrome induced by bee sting. Further investigations revealed grossly raised IgE with value of 338 IU/ml (reference value: < 150 IU/ml). Serum IgG was reported to be <177 mg/dl (reference value: 680-1445 mg/dl). IgA and IgM were normal (206.24 mg/dl and 151.52 mg/dl respectively).

The child was put on high protein diet with salt and fluid restriction, and was started on Prednisolone in a dose of 2 mg/kg/day. He showed rapid response to the treatment as evident by increased urine output and rapid decline in oedema. By seventh day of ensuing the therapy, proteinuria declined to 1+ on three successive days thus confirming the remission and so the patient was discharged. On follow up after one week patient was still in remission, and on one month follow up he continued to be in remission with nil urinary protein estimation and no oedema on clinical examination.

Discussion

Every year a rise in number of bee stings (members of order Hymenoptera) occurs during the flowering season. The manifestations vary from mild pain with local redness and swelling to massive oedema and anaphylaxis. The delayed/ late reaction with renal involvement in the form of Nephrotic syndrome has also been reported. The relationship between bee stings and Nephrotic syndrome have been well documented in the literature. The onset of clinical symptoms of Nephrotic syndrome is reported to appear between 5-7 days. Other manifestations reported in the literature are myocardial involvement, liver necrosis, CNS involvement, encephalopathy, GB syndrome, vasculitis, DIC and thrombocytopenia.

The severity of the manifestations of organ involvement, though dependent on the number of the bee stings, have also been reported following a single sting.⁴ In our patient, the manifestations occurred following a single bee sting, cause and effect relationship between Nephrotic syndrome and the bee sting was made on the basis of temporal association. The rapid onset of diuresis leading to decline in oedema and decrease in proteinurea following high dose of oral steroids observed in our patient has also been reported in similar profile of patients in the literature.⁴ Tauk B et al had reported poor response to corticosteroids and the use of cyclophosphamide to induce remission in patient who had developed MPGN following multiple wasp stings⁵

Bee venom mediated immunological response with involvement of T-lymphocyte and cytokine have been shown to affect glomerular basal membrane permeability, leading to proteinuria.³ The histopathological changes have been reported to be diverse, ranging from minimal change, mesangial proliferative glomerulonephritis, membranous glomerulonephritis and glomerulosclerosis.⁴ In our patient, good response to steroid leading to rapid onset of remission, the invasive renal biopsy to study histopathology of renal involvement was thwarted.

The manifestations of bee sting are primarily IgE mediated. In our patient, IgE levels were 338 IU/ml. Bee sting and allergens have been implicated in triggering Nephrotic syndrome. Also, relapse of Nephrotic syndrome have been reported after bee sting. However, preventing the relapse by blocking the specific allergic agent is not well supported by the literature.

What is new in this case: This case is a reminder of renal complications of bee sting, which can be successfully treated with steroid therapy. At the time of onset of Nephrotic syndrome, patient had already recovered from the manifestation of bee sting, but this case illustrates value of good history taking.

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