CASE REPORT

Acute Renal Failure Following Multiple Wasp Stings

Ghias Butt, Kifayat Ullah, Kinza Kanwal and Mudassir

Abstract

Wasp bite induced ATN from direct venom toxicity is very rare. We report two such cases. The first case was a 14 years old boy admitted with oliguria following multiple wasp stings. He had grossly deranged renal function requiring hemodialysis support. The other patient was a 24 years old man admitted with similar history and also required hemodialysis support. Renal biopsy in both cases was consistent with acute tubular necrosis without any casts or other changes, suggesting direct venom toxicity. Both the patients recovered completely after a period of few weeks, highlighting the importance of early detection and treatment of renal failure from wasp venom.

Key Words: *Wasp bite. Acute tubular necrosis. Wasp venom. Toxicity. Oliguria. Acute renal failure.*

INTRODUCTION

Renal failure can follow wasp bites but is usually not common. Wasp bite can cause renal failure as Acute Tubular Necrosis (ATN) and acute interstitial nephritis. Massive envomation can result in multi-organ dysfunction with renal, hepatic cardiac and neuronal toxicity.[1,2] AIN is thought to be due to delayed type immune complex mediated hypersensitivity reaction.[3] ATN following wasp bite can result from rhabdomyolysis, hemolysis or hypotension.[4] ATN following direct venom toxicity of wasps is very rare.[5]

We hereby present two unusual cases of renal failure secondary to direct venom toxicity.

CASE REPORT

Case 1: A 14 years old healthy boy presented with burning pain all over scalp, face, arms, back and legs following wasp stings. He was playing with other children in the village, when a nest of wasps fell upon him from a tree. The wasps were around thirty in number according to the witnesses. He was then treated by a local doctor for 3 days before referral to our center. He noticed dark color of his urine for around 3 days following the incidence but the amount of urine was reported normal. He started experiencing multiple episodes of non-projectile vomiting upon food ingestion after the incident.

He was refered to our center after 5 days of the incident due to persistent vomiting. Past medical or surgical history was unremarkable. Clinical examination showed blood pressure of 130/70 mm of Hg, a heart rate of 90/minute, and swelling and redness on scalp, back, legs and face. There were multiple sting marks over the head, back, both legs and face. Rest of the examination was normal.

Laboratory investigation showed a serum creatinine of 12.8 mg/dl, blood urea of 410 mg/dl, Hematocrit was 26.6 with neutrophil leukocytosis (14000) and platelet count was 222000/ul. The urine was yellow and showed proteinuria of +2 and numerous RBCS. PT and APTT were normal. CPK level was 995 U/L and CKMB level was 29 U/L. Bilirubin level was 0.44 mg/dl, ALT was 221 u/l; alkaline phosphatase level was 217 U/L, calcium level was 7.60 mg/dl, LDH level was 1548 mu/l, uric acid level was 6 mg/dl and phosphorus level was 10.98 mg/dl. Ultrasound of the abdomen showed enlarged kidneys with normal shape. There was no hydro-nephrosis or nephrolithiasis. Renal echogenicity was increased. As the muscle enzymes were mildly raised and liver enzymes were normal, rhabdomyolysis and hemolysis were ruled out. Peripheral blood film was also unremarkable.

The patient was started on intensive hemodialysis and continued with antibiotics and anti-histamines till the resolution of local inflammation. He also received intravenous steroids for possible acute interstitial nephritis to hasten the renal recovery. A renal biopsy was performed to confirm the renal lesion and the report was consistent with acute tubular necrosis but tubules were without any casts suggesting direct venom toxicity as the possible cause. A total of 5 sessions of hemodialysis were done, after which the dialysis was held and he was monitored for renal recovery. His kidney function improved over a period of 15 days with normalization of liver function and electrolytes.

Case 2: A 24 years old man from a hilly area, farmer by profession, admitted through emergency department with history of multiple wasp stings received on the farm. He received initial treatment within antihistamines and steroids at local hospital. He did not have any history of hypotension at that time. He started having vomiting and

Department of Nephrology, Pakistan Institute of Medical Sciences, Islamabad.

Correspondence: Dr. Kifayat Ullah, Department of Nephrology, Pakistan Institute of Medical Sciences, Islamabad.
E-mail: drkifayat@gmail.com

Received: December 29, 2013; Accepted: April 22, 2014.

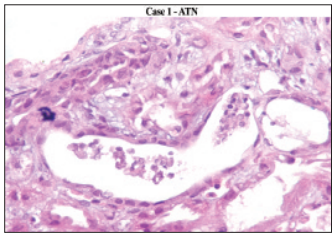

Figure 1: Renal biopsy showing acute tubular necrosis.

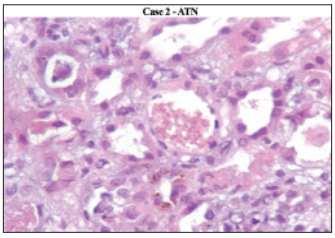

Figure 2: Renal biopsy showing acute tubular necrosis.

decreased urine output 5 days after the above event. He was admitted at local hospital for 3 days where he was initiated hemodialysis and received 2 sessions there. He was then refered to our center for further management. Initial assessment in emergency showed around 30 wasp bites on both arms, face and back. His past medical and surgical history was unremarkable. He denied any history of NSAIDs or other drugs. He was a smoker with 5 pack year history.

On examination, he was having normal vitals. Systemic examination was remarkable only for multiple bites on both arms, scalp and back. His biochemistry was consistent with marked renal dysfunction, with no evidence of hemolysis or rhabdomyolysis. Kidneys were normal in size and had grade 1 renal parenchymal changes. He was admitted and received supportive treatment for suspected ATN. Renal biopsy was done and was consistent with ATN. The tubules did not contain any casts confirming the suspicion of renal injury due to direct venom toxicity. He required further 3 sessions of dialysis after which kidneys opened up and he regained normal urine output and renal function.

DISCUSSION

Wasp bite induced renal failure usually results from pigment induced renal damage secondary to rhabdomyolysis or hemolysis or acute interstitial nephritis.[4]

The manifestations other than renal failure include myocardial necrosis and infarction, centrilobular necrosis of liver, and thrombocytopenia as a result of direct platelet toxicity.[6] The cause of liver damage precisely is not known but direct venom toxicity is suggested. Thrombocytopenia results from direct platelet toxicity. Rhabdomyolysis results from actions of toxin components phospholipase, polypeptides, histamine and serotonin. Intravascular hemolysis results from phospholipase and basic proteins.[4,6]

Other possible causes of renal failure include acute interstitial nephritis and tubular necrosis secondary to direct venom toxicity. Renal biopsy from such cases usually show tubular necrosis with pigments casts in the lumen.

Nace *et al*. have reported a case of acute renal failure without rhabdomyolysis and hemolysis suggesting direct venom toxicity as the cause.[5]

These cases are unique in the sense that both the cases were consistent with tubular necrosis but without any evidence of hemolysis or rhabdomyolysis clinically as well as biochemically, suggesting direct venom toxicity as the probable cause of renal failure.

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