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ESRD (End Stage Renal Disease) - A Rare Complication of Snake Bite Envenomation In Tropics - A Case Report

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Abstract
Snake bite is a well known occupational hazard amongst farmers, plantation workers, and other outdoor workers and results in much morbidity and mortality throughout the world. India is estimated to have the highest snake bite mortality in the world with almost 83,000 snake bites per annum with 11,000 deaths. Most of the fatalities are due to victim not reaching the hospital on time. Here by presenting you case of snake bite who came after 2 days of the bite with coagulapathy and acute renal failure, was given 6 vials of anti snake venom and was put on dialysis because of the worsening kidney dysfunction.

Case Report

The patient Mrs. A, a 64 years old healthy female with no previous known co-morbidities, was bitten by a snake on her left foot at around 4:30 pm while working in the paddy fields. The snake was caught and was killed, later identified as Viper. Following this she developed left lower limb pain with swelling. She was immediately rushed to a local hospital where she was reassured, treated conservatively and was sent back home. The very next day she developed red coloured urine, and the swelling and the pain were progressive. Hence she was again taken to the same hospital where again she was reassured, treated conservatively and was sent back home. After 2 days of the bite, she developed decrease urine output. In view of worsening pain and swelling, and decrease urine output with occasional hematuria, she was brought to our centre for further management.

On arrival in the ER, she was conscious, oriented with GCS of 15/15. Her vitals were stable. Systemic examination was unremarkable. On local examination, there was left sided pedal oedema, with tenderness and redness of the skin. There was no erysipelas. 20 minutes whole blood clotting time was negative. Initial laboratory investigations revealed haemoglobin of 9.4 dm/dL, platelets of 11,000 cells/mm3, BUN of 65 mg/dL, Creatinine 5.5 mg/dL, INR of 1.14, with all other values within normal limits. Peripheral Smear revealed microcytic hypochromic anaemia

with schistocytes and thrombocytopenia. Urine myoglobin was positive and serum LDH was 2810 U/L with CPK of 601 U/L and Fibrinogen of 139 mg/dl. With this a working diagnosis of coagulopathy and thrombotic microangiopathy secondary to snake bite causing acute renal failure was made. 6 vials of ASV were administered and then she was shifted to ICU for further management.

Nephrology opinion was sought and patient was dialysed. She underwent total 5 cycles of hemodialysis. Serial monitoring of CBC and RFT were done. The patient made a remarkable recovery as her cellulitis settled, her urine output improved with repeat RFT on the day of discharge was 2.9 mg/dL. Patient was discharged after 10 days of hospitalisation.

Discussion

Snake bite is a significant public health problem causing considerable morbidity and mortality worldwide, particularly in tropics. According to WHO, about 5 million people are bitten each year by poisonous snakes which results in 2.5 million envenomations, at least 100000 deaths, and 300000 amputations and other permanent disabilities. India is estimated to have the highest snake bite mortality in the world with almost 83,000 snake bites per annum with 11,000 deaths. Acute kidney injury (AKI) is an important complication of snake bite and a major cause of mortality. AKI is common after bites from myotoxic or hemotoxic snakes. The onset of renal failure varies from a few hours to as late as 96 hours after the bite[1]. Renal pathologic changes include tubular necrosis, cortical necrosis, interstitial nephritis, glomerulonephritis, and vasculitis. Hemodynamic alterations caused by vasoactive mediators and cytokines and direct nephrotoxicity account significantly for the development of nephropathy. Haemorrhage, hypotension, disseminated intravascular coagulation (DIC), intravascular hemolysis, and rhabdomyolysis enhance renal ischemia leading to AKI. Tubular necrosis is an important pathological correlate of AKI. Prolonged AKI with oligoanuria after snake bite is indicative of cortical necrosis or acute tubular necrosis associated with interstitial nephritis or extra capillary glomerulonephritis. Our patient probably developed renal cortical necrosis because of the longer course of the illness and recovery. Ideally a renal biopsy should have been done which could have helped in labelling the exact stage of the disease. Patients like these are highly susceptible of developing end stage renal disease. Since it is a reversible cause of chronic kidney disease, close monitoring of these patients should be done.

For the management, anti venom therapy is the key to the medical management of snakebite. The literature and the manufacturers of the snake antivenom recommend its early administration; within 6 hours in case of appearance of any local or systemic signs after snakebite, repeated every 4 to 6 hours until definitive improvement of the signs[2]. If compartment syndrome develops later (usually 52 hours after envenomations). However, in third world countries, time to treat generally exceeds 24 hours. An evidence-based study confirmed the empirical concept that a delayed time to treat should in no way exclude the use of antivenin immunotherapy in the case of African Vieira bites. Delayed administration of antivenin after 2 days, as in our case, proved to be beneficial in preventing the mortality. To conclude, antivenin administration should be considered in patients with envenomations complicated by marked and progressive local signs, delayed systemic signs and laboratory abnormalities more than 24 h after envenomation despite administration of earlier dose.

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