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Case report of snake bite during pregnancy

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ABSTRACT –

Snake bite in pregnancy can lead to significant maternal morbidity and mortality with poor fetal outcome. Here we report a primigravida aged 29 years at 23^{+3} weeks of gestation with viper bite with poor fetal outcome.

Keywords: acute kidney injury, anti-snake venom (ASV), coagulopathy, pregnancy, snake bite, dialysis

INTRODUCTION –

In hilly areas, the incidence of snake bites is common among the population. With over 270 species of snakes have been identified in India. Of these, around 52 are poisonous. Poisonous species of snakes are classified under 3 major families, i.e. Elapidae (common cobra, king cobra, krait), Viperidae (russell's viper, saw scaled viper, pit viper) and Hydrophiidae (sea snakes). Elapidae group is mainly neurotoxic, Viperidae are hematotoxic and Hydrophiidae are myotoxic. The delay in hospitalization and delay in initiating the accurate treatment is associated with high rate of morbidity and mortality. Here is a case of a pregnant female who presented to us with a snake bite and was treated with anti-snake venom (ASV).

CASE REPORT –

A 29-year old primigravida pregnant female with gestation period of 23^{+3} weeks was admitted to the emergency ward with presentation of localized swelling over the dorsum of left hand following snake bite while she was working in the garden with her husband. She was brought to the hospital on second day following the bite.

On presentation, she was conscious and oriented. She was hemodynamically stable with a blood pressure of 106/56 mm Hg, heart rate of 114 beats/minute, respiratory rate of 18/min and oxygen saturation of 96% on room air. Fang marks were visible over the dorsum of left hand with no active bleeding. There was progressive swelling of the left forearm with associated redness and tenderness with mild local increase in temperature, suggestive of cellulitis of left hand and forearm. She had developed hematuria and decreased urine output. Other systemic examination was within normal limit. There was no history of bleeding gums, hemoptysis, hematemesis, melena, vaginal bleed or epistaxis.

Investigations were as follows: Her complete blood count showed hemoglobin of 7.8 gm/dl, total counts of 29000/mm^3 and platelet count of 90,000/mm^3. Her renal functions were deranged with urea of 68 mg/dl and creatinine of 4.4 mg/dl suggestive of acute kidney injury (AKI). Liver functions showed total bilirubin of 0.50 mg%, direct bilirubin of 0.13mg%, AST of 729 IU/ml, ALT of 534 IU/ml, total proteins of 5.7 gm%

and albumin of 3.1 gm%. Her 20minute whole blood clotting test (WBCT) was >20 minutes suggestive of venom-induced consumption coagulopathy as blood did not clot. Serum electrolytes were normal. Urine microscopy showed numerous RBC but urine myoglobin was negative. Urine albumin was present. Electrocardiogram was normal. Ultrasound pelvis was done which showed absent fetal cardiac activity suggestive of intrauterine fetal death.

Patient was diagnosed as snake envenomation and AKI following snake bite. She was administered 100 ml (10 vials) of polyvalent anti-snake venom (ASV) in 200 ml of normal saline as an intravenous infusion over one hour. She did not develop any anaphylactic reaction following administration of ASV. Inj. Piperacillin was started for treating cellulitis. 20 min WBCT was repeated 6 hours after ASV administration and was normal. The next day, her vitals started stabilizing. On day 3rd, patient spontaneously aborted a male fetus. Inj. Oxytocin 10 IU was given in 500 ml of NS, following which uterus became well contracted with no active bleeding per vaginally. Patient's renal function tests were on increasing trend and urine output was also decreased. For this, she received 3 cycles of hemodialysis. Renal functions started improving and normalized in next 2 weeks. After full recovery, the patient was discharged on day 15th of admission after complete normalization of vitals, cellulitis and blood reports.

DISCUSSION –

Snake bite is a serious problem in tropics causing mortality worldwide every year.

Snake bites are considered as medical emergencies, affecting mainly the rural population. The snake venom comprises of variety of toxins which include enzymes, non-enzymatic polypeptide toxins and non-toxic proteins. The non-protein components are carbohydrates, lipids, metals and biogenic amines. Enzymes like phospholipase A2 damage mitochondria, RBC, WBC, platelets, peripheral nerve endings, skeletal muscles and vascular endothelium. Proteolytic enzymes cause local changes like blisters, edema and bruising. The neurotoxins are both presynaptic and postsynaptic. Death can occur due to bulbar and respiratory paralysis.[1]

Haemostatic abnormalities include spontaneous systemic hemorrhage mainly in the gingival sulci, hematuria, intracranial, subconjunctival and gastrointestinal hemorrhage. Other features include intravascular hemolysis, circulatory shock and renal failure secondary to acute tubular necrosis.[2]

Neurological symptoms following snake bite are commonly experienced within 6 hours. The symptoms include ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and neck muscle weakness (7.1%).[3] Encephalopathy is a rare presentation. Recovery is usually seen within a few hours to several days of ASV administration.[4]

A few patients develop AKI after getting bitten by venomous snakes, primarily vipers. Factors contributing to development of AKI include hemorrhage, hypotension, disseminated intravascular coagulation, and rhabdomyolysis. Enzymatic activities of snake venoms account for direct nephrotoxicity. Immunologic mechanism plays a minor role.

Early administration of ASV is an important therapeutic measure. ASV is recommended when a patient with snake bite develops one or more signs of systemic envenomation-hemostatic abnormalities: active bleeding, coagulopathy, or thrombocytopenia (<100 x 10^3/ml); neurotoxic signs like ptosis, external ophthalmoplegia, paralysis etc. ; cardiovascular abnormalities: hypotension, shock, arrhythmias, or abnormal ECG; AKI: oliguria/anuria, rising blood urea, and creatinine (>2mg/dl); hemoglobinuria/myoglobinuria; or local envenomation- local swelling involving more than half of the affected limb, swelling on the digits; and development of lymphadenopathy draining the affected limb.[5,6] Treatment stays the same after snake bite in pregnant females as for other victims of snake bite.[7] Overall rate of fetal loss is around 20% and maternal fatality rate is about 4-5%.[8]

The ASV available in India is polyvalent and is effective against four common species (Russell's viper, common cobra, common krait and saw-scaled viper). 8-10 vials of ASV are given for snake envenomation. The 20 WBCT is to be repeated only after 6 hours of ASV initiation because of inability of liver to replace clotting factors in less than 6 hours. 5-10 additional vials of ASV are administered if WBCT is more than 20 minutes. Local administration of ASV near bite site is ineffective and can lead to compartment syndrome. Treatment of AKI includes renal replacement therapy, fluids and supportive therapy. Mortality in snake bite-induced AKI is 1-20%.[9]

Delayed neurological manifestations may be seen with viper bites in some cases in the form of respiratory paralysis, despite receiving ASV. Henceforth, following snake bite, monitoring of all the patients should be done for at least 1 week in view of delayed neurological effect associated with ASV administration.[10]

CONCLUSION –

Patients presenting with a history of unusual bite and a local swelling with local signs of inflammation should be suspected as a snake bite. Clinical examination is crucial for assessment and making accurate diagnosis. ASV should be promptly administered in snake bite patient for a favourable outcome.

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