Case Report

Anaesthetic Management of Snake Bite Envenomation Complicated with Cellulitis for Fasciotomy

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Introduction

Snake bite envenomation is a life threatening emergency. Cellulitis at the local wound site of bite leads to compartment syndrome which requires urgent surgical intervention and fasciotomy. Though there are many reviews for medical management of snake bite, reviews for anaesthetic implications are scarce[1]. India is estimated to have the highest incidence of snakebite envenomation and mortality in the world[2]. The common poisonous snakes in India are cobra [*Naja naja*], Russell's viper [*Dabiola russelii*], saw-scaled viper [*Echis carinatus*] and common krait[*Bungarus caeruleus*][3]. In our patient there was envenomation by a species [*Naja naja*] commonly known as cobra. It belongs to family elapidae. The patient presented with neuromuscular paralysis, consumption coagulopathy, rhabdomyolysis and cellulitis with compartment syndrome requiring fasciotomy.

Case Report

58 year old female, with no known co-morbid illness was brought with alleged history of snake bite in left dorsum of foot. Snake was identified as cobra. The patient was initially treated at a local hospital where she was evaluated to have generalised muscle weakness associated with respiratory distress, which necessitated endotracheal intubation and shifted to our hospital with AMBU ventilation.

On arrival to casualty, the patient was drowsy, moving all four limbs, bilateral pupils were reacting to light and bilateral ptosis was present. Her blood pressure was 160/90 mm of Hg, pulse rate was 98/min, saturation was maintained at 98%. Endotracheal tube was in situ and she was ventilated with AMBU bag. Arterial blood gas analysis revealed respiratory acidosis. Patient was started on ringer's lactate and foley's catheterisation done. She was immediately shifted to intensive care unit and connected to ventilatory support (volume control with pressure support: Tidal volume-400ml, pressure support-10 cm of H_2O, positive end expiratory pressure-5 cms of H_2O, respiratory rate-12/min, Fraction of inspired concentration of O_2 - 0.5 to maintain SpO_2 of 100%). She was treated with intravenous crystalloids, antibiotics. Anti-snake venom(ASV) was started (10 vials in 500 ml of NS) as a slow i.v infusion. There were no allergic reactions. Her blood investigations like complete blood count, blood urea nitrogen, serum creatinine, bleeding and clotting time were within normal limits. Her creatinine kinase (CK) levels was 378 IU/L. INR was 1.38.

She responded well to the treatment and was weaned off from ventilator support and extubated on the next day. Her respiratory efforts were adequate and she maintained SPO_2 - 96% in room air. On day 2 of ICU CK and CK-MB showed increasing trend (Creatinine Kinase - 662 IU/L , Creatinine Kinase-MB = 149 IU/L). Whole Blood Clotting Time (WBCT) was positive > 20 minutes. She was given 10 vials of ASV infusion. On third day of intensive care, the patient developed cellulitis with compartment syndrome in her left leg. She was taken up for emergency fasciotomy under American Society of Anaesthesiologists (ASA) grade IIIE.

Preoperatively, she was conscious and oriented. Blood pressure, heart rate and respiratory rate were within normal limits. Her motor power was 3/5, in both upper and lower limbs. After shifting to operating table, her heart rate was 88/min, blood pressure was 140/90 mm Hg, electro cardiogram showed normal sinus rhythm. Intravenous Ringer's Lactate infusion was started at 100 ml/hr. Regional anaesthesia was planned. Patient was given Inj. fentanyl 75 mcg i.v. Left Sciatic and femoral nerve block using Braun Stimuplex nerve stimulator, (22G, 15 cms needle) was done. Drug prepared was 15 ml of 0.75% ropivacaine, 15 ml of 2% lignocaine with 1:200000 adrenaline and 15 ml NS. Left sciatic nerve block - Classic Labat's approach in semi-prone position. Dorsiflexion of ankle joint was obtained at 0.5 mA and it disappeared on reducing the current to 0.2 mA. 30 ml of drug given. Left femoral nerve block was done in supine position, patellar jerk was obtained at 0.5mA and it disappeared after reducing the current to 0.2 mA - 15 ml of the drug given. Onset of action was 8 minutes. Complete motor blockade was achieved in 14 minutes. Duration of the surgery was 30 minutes. Duration of sensory blockade was for 7 hours. Intra operative period was uneventful. Post operatively she was

shifted to intensive care unit and continued with antibiotics, analgesics and intravenous crystalloids.

On day 4 in intensive care unit patient's whole blood clotting time (WBCT) was still positive (more than 20 minutes), 5 vials of anti-snake venom infusion was repeated. Patient condition improved and she was shifted to the ward after 5 days in intensive care unit. She was discharged from the hospital in a stable condition with follow up for dressing of the wound.

Discussion

The cobra venom is a complex mixture of toxins and enzymes, each of which may be responsible for one or more distinct toxic actions[3,4].

Phospholipase A2 damages mitochondria, red blood cells, leucocytes, platelets, peripheral nerve endings, skeletal muscle, vascular endothelium, and other membranes, produces presynaptic neurotoxic activity, opiate-like sedative effects and leads to the release of histamine[3,4]. Hyaluronidase promotes the rapid spread of venom through tissues[3].

The venom also includes postsynaptic α-neurotoxins such as α-bungarotoxin and cobrotoxin. They bind to acetylcholine receptors at the motor endplate[5,6]. Progressive descending paralysis is the hallmark of systemic envenoming by elapid snakes. The early signs are bilateral ptosis, inability to protrude tongue and difficulty in speech. Limb weakness, loss of deep tendon reflexes, and fixed dilated pupils may follow[3,4].

Once paralysis reaches the diaphragm and the intercostal muscles, victims usually die of respiratory failure if they are not adequately ventilated[7]. Acetylcholinesterase, though found in most elapid venoms, does not contribute to their neurotoxicity[3,4]. Zinc metalloproteinase haemorrhagins causes damage to vascular endothelium, causing bleeding. This causes clotting disturbances because most of the fibrin clot is broken down immediately ("consumption coagulopathy").

This can be diagnosed by doing a 20 minute whole blood clotting time test[3]. Proteolytic enzymes (metalloproteinases, endopeptidases or hydrolases) and polypetide cytotoxins (cardiotoxins) increase vascular permeability causing oedema, blistering, bruising and necrosis at the site of the bite[3,5]. It also contains myotoxins like the phospholipase A2 which causes rhabdomyolysis. This leads to myoglobinuria and blockage of renal nephrules leading to acute tubular necrosis and acute renal failure[3,8,9].

Clinical features of compartment syndrome includes disproportionately severe pain, weakness of intracompartmental muscles, hypoaesthesia and obvious tenderness of the compartment on palpation. Detection of arterial pulses by palpation or doppler ultrasound probes, does not exclude intracompartmental ischaemia. Intracompartmental pressures exceeding 35 mmHg may carry a risk of ischaemic necrosis requiring fasciotomy[10]. Corticosteroids are not effective in ameliorating local effects of envenoming[11].

Problems	Expected complication	Precaution / Intervention	Monitoring
Myoglobinuria	Acute kidney injury	Avoid hypovolumia, hypotension, acidosis	Urine output, urine colour
Abnormal coagulation	Severe bleeding, DIC	Avoid neuraxial anaesthesia. Arrange adequate blood products	Coagulation tests at regular intervals, Clinical monitoring
Pain	Tachycardia, increased oxygen demand, release of inflammatory mediators	Adequate analgesics from the start	Clinical
Anemia (secondary to hemoglobinuria)	Tachycardia, decreased oxygen carrying capacity	Supplemental oxygen administration, blood transfusion	SpO_2, PaO_2, Arterial blood gas analysis
Direct effect on neuro muscular junction	Prolonged effects of neuro muscular blockers	Avoid neuro muscular blockers / use with caution with ventilator backup	Train of four monitoring
Direct myocardial toxicity	Cardiogenic shock, Myocardial infarction	Inotropic support, Supplemental oxygen administration	ECG, Echocardiogram, Cardiac enzymes,invasive arterial BP, CVP
Residual bulbar palsy, ineffective cough, inadequate respiratory efforts	Pulmomary aspiration, respiratory acidosis	Patient position, breathing exercises, ventilator backup	
Cellulitis	Compartment syndrome, polymicrobial infection, sepsis	Broad spectrum antibiotics, Culture from surgical site, fluid management (Surviving Sepsis Guidelines)	Clinical monitoring

Table 1 - Anaesthetic considerations of snake bite envenomation

Anaesthesia considerations

The anaesthesia for fasciotomy can be performed under general anaesthesia with antibiotic cover with neuro muscular blockade monitoring. The prolonged neuromuscular blockade with non depolarising muscle relaxant are produced by an additive or synergistic effect between the clinical neuromuscular blocker and the postsynaptic neurotoxin. There can be increased neuromuscular blockade after neostigmine, it could be due to anti-acetylcholinesterase toxicity, a venom-neostigmine interaction[12]. Postoperatively patient may require ventilatory support due to neuroparalytic respiratory failure[13].

Alternatively regional anaesthesia can be performed. However with active consumption coagulopathy central neuraxial blockade cannot be performed due to the risk of spinal hematoma[14]. Peripheral nerve blockade can be the anaesthesia of choice in case of coagulopathy or systemic toxicity effects. Preferably performed under ultrasound guided approach to minimise unexpected vascular injuries. Peripheral nerve blockade has fewer cardiac (hemodynamic) and respiratory effects than the other modes of anaesthesia and thus it can be the choice of anaesthesia in case of high risk patients like the one reported here. The problems, expected complication, intervention and monitoring are summarised in table -1.[7,8,9,13,14]

Conclusion

Complications due to snake bite envenomation are still increasing inspite of improved first aid strategies. Patients developing compartment syndrome are also high. There are various systemic effects due to snake venom that can complicate while administering anaesthesia to these patients posses a significant risk. Knowledge on the various systemic effects of envenomation and the physiological changes that follows, helps in framing a better choice of anaesthesia. Administering general anaesthesia or a central neuraxial blockade can worsen the morbidity of the envenomation. Hence the most safe option for anaesthesia in these patients is peripheral nerve blockade.

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