

RESPIRATORY REHABILITATION IN ACUTE CARE OF PATIENTS WITH NEUROPARALYTIC SNAKE ENVENOMATION: CASE SERIES

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ABSTRACT

Background: Snakebite is an environmental hazard associated with significant morbidity and mortality. Neurotoxic envenomations have the potency to cause a broad spectrum of presentations starting from ptosis and ophthalmoplegia to respiratory arrest. These patients require ventilatory assistance in addition to administration of anti-snake venom (ASV) and other supportive measures. Mechanically ventilated patients are at risk for retained secretions due to endotracheal intubation disrupting mucociliary escalator, relative immobility of mechanically patient confined to bed can lead to postoperative atelectasis, impaired cough, and retained secretions and thereby physical therapy may be indicated for patients in the intensive care setting.

Materials and Methods: A total of twenty four consecutive patients ranging in age from 25-45 years, who required, mechanical ventilation for respiratory muscle paralysis, secondary to snake envenomation, seen during three months period, recruited from various ICU's were included in the study. All the patients included were mechanically ventilated on Hamilton Evita ventilator, on volume control (CMV) mode with PEEP<10 cmH₂O and had stable hemodynamics with heart rate = 60-100 beats/min; MABP = 70-110mm Hg. Patients received chest physiotherapy intervention twice in a day. Effects of physiotherapy treatment were studied on static lung compliance (C_{ST}), oxygenation ratio (PaO₂:FiO₂ ratio), partial pressure of carbon dioxide in arterial blood (PaCO₂), cologarithm of activity of dissolved hydrogen ions in arterial blood (pH) and chest X-rays. Measurements of dependent variables were recorded (PRE) before commencement of treatment, 30 minutes and 60 minutes after treatment. Physiotherapy intervention included bronchial hygiene therapy and manual hyperinflation using Mapleson-C circuit.

Results: Analysis of variance showed that there was highly significant improvement in C_{ST} mean values (p<0.01) and significant improvement in PaCO₂ mean values (p<0.05). However, pH and PaO₂:FiO₂ ratio could not reach statistical significance. Comparing mean values with critical difference, significant critical difference was observed between mean values at PRE and Post-30, and between PRE and Post-60 time intervals (p<0.05) for C_{ST}, PaO₂:FiO₂ ratio and PaCO₂, respectively.

Conclusion: Respiratory rehabilitation of patients with neuroparalytic snake envenomation can be effectively achieved with employment of various physiotherapeutic techniques including manual hyperinflation and bronchial hygiene therapy.

KEYWORDS: Respiratory Rehabilitation, Neuroparalytic Snake Envenomation, Bronchial Hygiene therapy.

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INTRODUCTION

Snakebite is an environmental hazard associated with significant morbidity and mortality [1].

Snakes bite millions of people annually, creating one of the neglected health problems of the tropics. Snake bites remain a public health

problem in many countries even though it is difficult to be precise about the actual number of cases [2]. It is estimated that more than five million bites by venomous snakes occur worldwide, with 1,25,000 deaths annually. It is a common medical emergency encountered in South Asia [3]. The largest numbers of fatal snakebites occur in South Asia and Africa. [4] In Asia alone, snakebites are estimated at about four million cases per year, of which approximately 50% are envenomations, with about 1,00,000 annual deaths. In India, an estimated 2,00,000 persons per year fall prey to snake-bite, with an estimated fatality rate of 35,000-50,000 per year [1].

We present our experience with physical therapy management of patients in various Intensive Care Units (ICU's) of the hospital with severe neuromuscular manifestations and acute ventilatory failure.

MATERIALS AND METHODS

A total of twenty four consecutive patients ranging in age from 25-45 years, who required, mechanical ventilation for respiratory muscle paralysis, secondary to snake envenomation, seen during three months period, were recruited from various ICU's in the institute and included in the study. All the patients included were mechanically ventilated on Hamilton Evita ventilator, on volume control (CMV) mode with PEEP < 10 cmH₂O and had stable hemodynamics with heart rate = 60-100 beats/min; MABP = 70-110 mm Hg. Snake bite victims with mild neuromuscular weakness not requiring ventilatory assistance, and also patients with systemic manifestations other than neuromuscular paralysis, were excluded from the study. Patients suffering with acute cardiac dysarrhythmias/ added cardiac pathology, undrained pneumothorax, obstructive lung disease, haemoptysis of unknown cause, severe bronchospasm and any associated head injuries, chest wall deformity, history of smoking and impaired lung mechanics were excluded from the study.

Detailed history and physical examination, including the onset and nature of symptoms, site of bite, local reaction at the bite site and general and systemic manifestations, laboratory investigations performed at admission included

arterial blood gas (ABG) analysis, electrocardiogram (ECG), chest radiograph, serum biochemistry, haemogram and coagulogram, renal function were recorded for each patient. All patients were managed with wound care at bite site, tetanus prophylaxis, anti-snake venom and appropriate antibiotics instituted.

All the patients were ventilated initially in volume controlled mode. With the improvement of neuromuscular paralysis, weaning was implemented through SIMV-CPAP modes. When successfully tolerated, they were put on T-piece trial. Extubation was done after clinically assessment and monitoring of arterial blood gas parameters by the medical personnel's. All patients received chest physiotherapy with maintenance of asepsis, and care of ETT. The patients were observed for evidence of side effects of ASV, ventilator associated complications such as pneumonia, septicemia or barotrauma.

Procedure: All the patients who met the inclusion criteria were evaluated thoroughly using an evaluation performa. Patients received chest physiotherapy intervention twice in a day. The minimum washout interval between the two treatment sessions was three hours apart to allow the patients to recover and to minimize any carryover effect. Each treatment session was of 45-50 minutes duration. Effects of physiotherapy treatment were studied on static lung compliance (C_{ST}), oxygenation ($PaO_2:FiO_2$) partial pressure of carbon dioxide in arterial blood ($PaCO_2$), cologarithm of activity of dissolved hydrogen ions in arterial blood (pH) and chest X-ray's. Baseline measurements of dependent variables (PRE), and were obtained five-minutes before commencement of treatment. Chest X-rays were analyzed and reported by medical staff. During the entire procedure, all precautions were undertaken to prevent infection to the patients. Heart rate (HR), electrocardiogram, mean arterial blood pressure (MABP), arterial blood oxygen saturation (SpO_2) and temperature were monitored using bedside monitor (Philips Intelli Vue MP 40, Philips International B.V., The Netherlands).

Patients were placed in the indicated postural

drainage position for a time period of ten minutes, in accordance with the reporting of chest X-ray's. Bronchial hygiene therapy was then given to all patients followed by endotracheal suctioning to remove excess secretions. The FiO₂ of the ventilator was then set to 1.0 and the patients were left undisturbed for 15 minutes before the next intervention. Patients were then given recruitment maneuver in form of manual hyperinflation.

Manual hyperinflation: Manual hyperinflation was performed using Mapleson-"C" circuit and a two liter reservoir bag (Intersurgical Mapleson C circuit with two liter bag) connected to 100% wall oxygen at 15 L per minute. The waveform consisted of an inspiration of three seconds, sustained inspiration for 2 seconds, and a fast release of the valve to ensure a short expiration, during which bag was held compressed. Expiration was passive and unobstructed to facilitate expiratory flow with no PEEP applied. The I: E ratio was 2:1. A manometer (Medisys pressure manometer with T-piece connector) was included in the circuit and patients were manually hyperinflated to a maximum peak airway pressure of 40cm H₂O. With a two-handed technique, six sets of six MHI breaths were delivered to the patient. Each MHI set was followed by six tidal breaths to a peak airway pressure of 20cm H₂O. The patients were suctioned three times throughout the procedure following every second set of hyperinflation breaths and treatment was of 20 minutes duration.

Measurements: Post intervention measures of the dependent variables were recorded at 30 minutes (POST-30) and 60 minutes (POST-60) after the intervention. Static lung compliance (C_{ST}) readings were recorded from the display on the ventilator. An average of three readings of static pulmonary compliance was taken. Arterial blood gas analysis samples were taken to monitor oxygenation (PaO₂:FiO₂), partial pressure of carbon dioxide in arterial blood (PaCO₂) and cologarithm of activity of dissolved hydrogen ions in arterial blood (pH). Chest X-ray monitoring was done on daily basis. Heart rate (HR) and Mean arterial blood pressure (MABP) were read directly from the monitoring system and recorded before

intervention (for Manual Hyperinflation intervention recording was done after disconnection from the ventilator); 1-minute during intervention; and 1, 5, and 20 minutes following intervention.

Data analysis: The data collected for different variables at different time intervals was subjected to statistical analysis using SAS software (version 6.0, Chicago, IL). Mean and standard deviation of demographic variables were calculated for all patients. Measurements of different variables (C_{ST}, PaO₂/FiO₂ ratio, PaCO₂, pH, HR and MABP) before and after intervention were compared using One way Analysis of Variance, to test the effect of time interval on different variables and the time interval means were compared using critical difference (C.D.) values (p<0.05).

RESULTS

Table 1: Subject Characteristics [Mean ±S.D.].

Sr. No.	Variables	Mean ±S.D.
1	Age (yrs)	32.75 ±6.89
2	Tidal volume (ml/Kg)	6.73 ± 0.44
3	PEEP (cm H ₂ O)	5.75 ± 0.97
4	FiO ₂ (%)	0.38 ± 0.06

Table 2: Clinical and Laboratory Findings in Patients with Severe Neuroparalytic Snake Envenomation.

Symptoms and Signs	Number of Patients (%)
Ptosis	20(100%)
Pupillary Reaction(right and /or left; dilated and /or non reacting)	14(70%)
Dysphagia	10(50%)
Dysphonia	10(50%)
Drooling	10(50%)
Abdominal pain	9(45%)
Myalgia	10(50%)
Weakness	20(100%)
Extraocular signs(extraocular muscle palsy, ptosis, paresis and /or areflexia-superficial and deep reflex)	20(100%)
Chest pain and /or palpitation	0(0%)
Cyanosis	18(85%)
Dyspnoea	20(100%)
Tachypnoea	16(80%)
Laboratory Abnormalities	Number of Patients (%)
Hypercapnia	20(100%)
Hypoxaemia	10(50%)
Leukocytosis	10(50%)
Acidosis	9(45%)
Abnormal chest radiograph	10(50%)
Coagulopathy	0(0%)

Thirty victims, following snake bite envenomation by various snakes were admitted to various ICU's of this Institute during the study period, of which 24 patients (16 males and eight females) with subsequent neuromuscular respiratory failure ventilatory failure were given chest physiotherapy management. The age ranged between 25 to 45 years (Table 1). All the patients had severe neuromuscular manifestations (Table 2).

Analysis of variance showed that there was highly significant improvement in C_{ST} mean values ($p < 0.01$) and significant improvement in $PaCO_2$ mean values, at Post-30 and Post 60 time intervals ($p < 0.05$) (Table 3). However, pH and $PaO_2:FiO_2$ ratio could not reach statistical significance.

Table 3: Measurements of Dependent Variables before and after Physiotherapy Treatment at various intervals of time.

Variable	PRE (Before Intervention)	POST-30 (30 minutes after Intervention)	POST-60 (60 minutes after Intervention)	P value	F value	LSD
	(Mean \pm S.D.)					
pH	7.34 \pm 0.08 ^a	7.38 \pm 0.06 ^a (0.54%)	7.37 \pm 0.05 ^a -0.41%	0.424	0.89	0.0673
$PaO_2:FiO_2$ Ratio	218.79 \pm 110.90 ^b	343.66 \pm 126.49 ^a (57.07%)	296.71 \pm 89.48 ^{ab} (35.61%)	0.096	2.63	114.39
$PaCO_2$ (mm Hg)	45.84 \pm 8.14 ^a	35.91 \pm 6.58 ^b (-21.66%)	38.55 \pm 6.07 ^b (-15.90%)	0.027 ^{**}	4.33	7.263
C_{stat} (mL/ cm H ₂ O)	38.74 \pm 2.31 ^b	43.55 \pm 2.82 ^a (12.41)	41.55 \pm 2.59 ^a (7.25%)	0.005 ^{***}	7	2.687

pH= power of hydrogen (cologarithm of the activity of dissolved hydrogen ions in arterial blood); $PaO_2:FiO_2$ = oxygenation; $PaCO_2$ = partial pressure of carbon dioxide in arterial blood ; C_{stat} = static lung compliance; S.D. = standard deviation.

*Values in parenthesis are mean percentage changes from the baseline measurements.

**Measurements are statistically significant at $P < 0.05$;

*** Highly Significant at $P < 0.01$.

Means with same superscripts in a column are not significantly different ($P < 0.05$), LSD = Least Significant Difference.

Comparing the mean values for pH at various intervals of time, there was percentage increase in means from baseline for pH mean values at Post-30 (0.54%) and Post-60 (0.41%) time interval. However, post hoc analysis of mean values revealed no significant critical difference between pH values at various intervals of time.

For $PaO_2:FiO_2$ ratio mean values, the percentage increase in means at Post-30 and Post-60 time interval was by 57.07% and 35.61%, respectively. Also, there was significant critical difference between mean values observed at PRE and Post-30, and between PRE and Post-60 time intervals ($p < 0.05$).

There was significant percentage reduction in $PaCO_2$ mean values at Post-30 (21.66%) and

Post-60 (15.90%) time intervals compared to baseline ($p < 0.05$). In addition, there was a significant critical difference between mean values observed at Post-30 and Post-60 time intervals ($p < 0.05$).

C_{ST} mean values were showed highly significant improvement at Post-30 and Post-60 time intervals after intervention ($p < 0.01$). Comparing C_{ST} mean values in terms of percentage improvement, there was significant percentage increase in C_{ST} mean values at Post-30 (12.41%) and Post-60 (7.25%) time intervals. In addition, post-hoc analysis revealed that means had significant critical difference between each other ($p < 0.05$). There were no adverse changes in blood pressure, heart rate, or heart rhythm.

Chest X-rays showed marked changes from baseline (Fig.1).

Fig.1a: Chest X-Ray (Before Intervention).



Fig.1b: Chest X-Ray (After Intervention).



DISCUSSION

The morbidity and mortality following snake bites is a frequently encountered problem in India. [8] In India, approximately 15,000 persons die of bites by poisonous snakes every year [9]. Delayed presentation to hospitals frequently contributes to increased morbidity and mortality from snakebites. The delay is due to people using folk and indigenous remedies before reaching the hospital especially in rural areas [5].

The victims of snake bites are mainly of the rural population, who are bitten during field work, farming communities and when sleeping outdoors.[4,6] Factors responsible for high

incidence of snake bite in Asia are ecological conditions i.e. temperate weather and socio-economic factors i.e. high population density, wide spread agricultural activities in rural areas, lack of functional snake bite control program and numerous venomous snake species [4]. Contributing to this in developing nations, there is also deficiencies in the management of complications, transportation, hospital equipments and public knowledge of appropriate first aid, which result in a mortality rate one hundred fold higher than in developed countries [7].

Poisonous species of snakes are included in five families: viperidae (vipers), elapidae (cobras), colubridae, atractaspididae and hydrophidae (sea snakes). Out of them vipers and cobras are common species in South East Asia [4]. Elapid snake bites are among the most lethal, resulting in muscle paralysis due to curare like neuromuscular blocking action affecting the muscles of eyes, throat and chest [8].

According to toxicity, snakes are categorized as hematotoxic, neurotoxic and myotoxic. Among the neurotoxic group, the majority of bites are due to *Naja naja* (common cobra), *Ophiophagus hannah* (king cobra) and *Bungarus caeruleus* (Krait) in India [9].

Snake venom is complex mixture of proteins and small polypeptides with enzymatic activity [7]. The snake venom consists of different enzymatic and non-enzymatic components loosely categorized as neurotoxins and hemorrhages [9]. Snake bites have two major effects locally the venom causes an intense inflammation and destroys the tissues by proteolytic enzymes the vascular walls, including edema, sometime over the large area, often with blood loss. The venom proteins, when in high concentration in the blood, cause shock, generalized symptoms such as hypotension and abdominal cramps, allergic sensation, coagulopathy and rarely death [4].

In our ICU's, most of the cases admitted are neurotoxic snakebites from the above mentioned three commonest varieties. Neurotoxic envenomations have the potency to cause a broad spectrum of presentations starting from ptosis and ophthalmoplegia to respiratory arrest [9]. The Bungarotoxin present in neuroparalytic snake venom acts on presynaptic and synaptic sites. The polyvalent anti snake venom, which

is effective in reversing synaptic blockade, cannot reverse the presynaptic block [8,10,11]. The systemic effects of venom of elapid snakes (cobra and krait) are predominantly neurotoxic, causing a selective neuromuscular block, affecting mainly the muscles of eyes, tongue, throat and chest leading to respiratory failure [10,11]. Severity of envenomations and respiratory paralysis is related to dose of venom injected, potency of venom, anatomic location of bite, age, health and immune status of the victim and timely medical intervention. These patients require ventilatory assistance in addition to administration of anti-snake venom (ASV) and other supportive measures [9,10]. Timely administered anti-snake venom and ventilatory assistance can prevent the mortality and morbidity of the victims [9].

Mechanically ventilated patients are at risk for retained secretions due to endotracheal intubation disrupting mucociliary escalator, relative immobility of mechanically patient confined to bed can lead to postoperative atelectasis, impaired cough, and retained secretions [12].

Physical therapy may be indicated for patients in the intensive care setting when they have retained secretions and radiological evidence of atelectasis or infiltrate, or as prophylaxis in conditions such as acute head injury and smoke inhalation. Physical therapy interventions include postural drainage, breathing exercises, percussion, vibration, manual hyperinflation, coughing, huffing, and suction. Body positioning, which primarily aims to optimize ventilation-perfusion ratios, and mobilization and exercise are physical therapy interventions not traditionally considered as part of the treatment for these patients [13].

C_{ST} is considered as important clinical outcome measure, and it may be used to predict mortality in patients with respiratory failure [14]. In the present study, there was a highly significant improvement in static lung compliance, 30 and 60 minutes after intervention. The application of manual hyperinflation with a larger than normal tidal volume breath coupled with an inspiratory pause adopted in this study, may have facilitated collateral ventilation and effective recruitment of alveoli, thereby improving the

time-dependent elastic behavior of the lung. There was also a possibility that the manual hyperinflation technique was effective in mobilisation of pulmonary secretions from peripheral to central airways, which were subsequently removed with suctioning, thereby leading to further recruitment of more functional alveolar units [15]. In addition, the immediate improvement in lung compliance with MHI could be attributed to the use of Mapleson-C circuit which produces significantly larger inspiratory pressures and tidal volumes than other circuits during manual hyperinflation [16].

Evidence exists to suggest that improvement in lung compliance after hyperinflation breaths occurs because of the re-expansion of previously collapsed lung units. When lung inflation is sustained for a few seconds, as in this study, permits a more uniform distribution of gas, recruiting interdependent collapsed alveoli through collateral ventilation or intercommunicating channels and consequently improving functional residual capacity (FRC) [17].

There was a significant reduction in PaCO₂ levels ($p < 0.05$). The reduction in PaCO₂ levels at Post-30 and Post-60 minute time interval could be attributed to the marked wash out of CO₂ from blood after intervention [17,18]. The pH changes showed percentage improvement, but could not reach statistical significance. This could be because the readings were taken on single day session and pH changes take time to recover [19]. In addition, PaO₂:FiO₂ ratio mean values showed percentage improvement at Post-30 and Post-60 time intervals and also there was a significant critical difference between the mean values. The probable reason is that with increase in the recruitment of functional alveolar units after intervention, there may have been an improvement in the ventilation-perfusion ratio; decreased shunting of blood in lungs and improved oxygen transport in the blood [18]. However, the ratio could not reach statistical significance. This could be explained on the basis of hypoxic effects of endotracheal suctioning after manual hyperinflation [20].

CONCLUSION

Respiratory rehabilitation of patients with neuroparalytic snake envenomation can be

effectively achieved with employment of various physiotherapeutic techniques including manual hyperinflation, postural drainage, percussion, vibration and chest expansion exercises.

ABBREVIATIONS

C_{st} - Static lung compliance

PaO_2 - FiO_2 ratio :-Oxygenation ratio

$PaCO_2$ - Partial pressure of carbon dioxide in arterial blood: -

pH - Cologarithm of activity of dissolved hydrogen ions in arterial blood: -

MHI - Manual Hyperinflation

Conflicts of interest: None

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