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Clinical Case Control Study of Injection Calcium Gluconate in Krait Snake Bite at Tertiary Care Centre in Tamilnadu

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ABSTRACT

In this study, to evaluate the effectiveness of calcium gluconate in early and complete recovery of krait snake bite admitted in government thiruvannamalai Medical College and Hospital. A prospective randomized clinical control study conducted in the Department of general medicine, Government thiruvannamalai Medical College and Hospital, thiruvannamalai. The study population are the Patients admitted with krait bite in Government thiruvannamalai Medical College and Hospital, thiruvannamalai and the study duration is 18 months and sample size of 100 patients. Results will be collected, tabulated and followed by statistically analyzation using Microsoft Excel and SPSS to detect the usefulness of calcium gluconate in krait bite. The response after calcium gluconate in the case and control group were compared. The higher number of complete recoveries was observed in <1 day of patients in case group. The p-value is 0.001 <0.05 statistically significant were observed. Krait bites with neuroparalytic symptoms may be effectively treated with intravenous calcium gluconate therapy in combination with anti-snake venom (ASV). Krait venom often induces presynaptic inhibition, where calcium functions as a neurotransmitter. We find that administering calcium gluconate infusion at an early stage in patients with Krait bite and neurotoxic envenomation results with reduced mortality and morbidity.

INTRODUCTION

The common krait's venom consists of powerful neurotoxins, which induce muscle paralysis. On analyzing we can see that this venom contains presynaptic and postsynaptic neurotoxins, which generally affect the nerve endings. Incidents occur mainly at night because kraits are nocturnal[1]. Frequently, little or no pain occurs from a krait bite (Krait's bites are significant for inducing minimal amounts of local inflammation/swelling) and this can give false assurance to the victim[2]. Typical symptoms after a krait-bite are, severe abdominal cramps, accompanied by progressive paralysis. As no local symptoms present, a patient should be carefully observed for signs of paralysis (the onset of ptosis) and treated with anti-snake venom. It's also possible to assist by mechanical ventilation at hospitals[3]. The few symptoms of having been bitten are: the facial muscles get tight in one to two hours, the patient may be unable to talk or see and, if left untreated, the patient may die from respiratory paralysis within four to five hours. A clinical toxicology study gives an untreated mortality rate of 70-80%. It is the most poisonous snake in India and its venom is presynaptic neurotoxic in nature[4]. ß-Bungarotoxins, which constitute greater than 20% of the protein content of krait venom, act presynaptically and are believed to be the most toxic components of the venom, usually causes presynaptic toxicity where calcium acts as a neurotransmitter., depolarization of neurons causes voltage gated ion channels to open into the cell which causes influx of calcium ions into the cell. Calcium helps in mobilizing the vesicles, which prepare them for neurotransmitter release[5,6]. Calcium interacts with synapsin a protein which helps in binding synaptic vesicles with cytoskeleton of the cell and causes synapsin to separate from the vesicles[7]. After mobilization synaptic vesicles goes and binds with presynaptic membrane with the help of a complex called snare complex. Synaptotagmin, a calcium sensor protein found in synaptic vesicles helps in fusion of vesicles to presynaptic membrane. After binding with membrane, the vesicles release its contents into cleft[8]. Beta bungarotoxins have phospholipase activity that hydrolyses phosphoglycerides, produce neuromuscular blockade by damaging it thus inhibiting the release of acetylcholine from the motor nerve terminals. These neurotoxins appear to have a triphasic effect on Ach release[9,10]. First, there is a decrease, followed by a transient increase and then a complete block of release. The initial two phases are reported to be independent of phospholipase A2 activity., however, the late phase is directly related to enzymatic activity.

MATERIALS AND METHODS

A prospective randomized clinical control study done at the Department of general medicine, Government Tiruvannamalai Medical College and Hospital., Tiruvannamalai in the Patients admitted with krait snake bite. The duration of study is 18 months.

The Sample Size is 100 and Allotted as Follows:

- **Group 1:** One group of people with administration of calcium gluconate (50 Patients).
- **Group 2:** One group of people without administration of calcium gluconate ((50 Patients).

Inclusion Criteria:

- Patient between the ages of 19-70 yrs.
- Patient with krait bite with neurotoxicity.
- Patient who are ready to give written informed consent.

Exclusion Criteria:

- Patient with other than krait bites.
- Patient with known neurological conditions.
- Patient with hypercalcemia.
- Patient with severe hypophosphatemia.
- Patient with hypersensitivity to calcium gluconate.
- Patient on treatment with cardiac glycosides.
- Patient who refuses to give consent.

Methodology: The patients selected are who fulfils the inclusion criteria and get consent and willingness to participate in the study.

- 100 krait bite patients will be selected out of which 50 patients were given with 10 ml of 10% calcium gluconate every 6 hours until recovery., the other 50 were kept as control.
- Results will be collected, tabulated, and followed by statistically analyzation using Microsoft Excel and SPSS to detect the usefulness of calcium gluconate in krait bite.

Data Analysis: Research data will be collected in the Data Proforma Sheet and saved in Excel. Statistical Analysis will be done by SPSS-23 software. The tests will be presented in descriptive statistics with 5% level of significance and 95%confidence interval.

Statistical Analysis: Statistical analysis done mean, standard deviation and p-value are made out. A p-value <0.05 (typically ≤0.05) is statistically significant and a p-value higher than 0.05 (>0.05) is not statistically significant.

RESULTS AND DISCUSSIONS

Comparison between the age distributions of the case and control groups were calculated and given in (Table 1). In all age groups, the almost equal number of patients was observed in both case and control group. The p-value is 0.444 (>0.05) statistically not significant were observed.

Table 1: Comparison Between the Age Distributions of the Case and Control Group

Age distribution	Case	Control
<20 Years	2	2
21-30 Years	3	10
31-40 Years	13	9
41-50 Years	13	11
51-60 Years	11	10
61-70 Years	8	8
P-Value	0.444	

The higher number of patients (80%) was admitted in hospital with <24 hours as shown in (Table 2). Very few patients were admitted in hospital with >5 days of bite was observed.

Table 2: Time of Bite to Arrival to Hospital of the Patients and their Percentages

Time of Bite to Arrival to Hospital	Number	%
<2 hours	31	31
6 hours	58	58
24 hours	11	11
TOTAL	100	100

Responses after giving early Inj. Calcium gluconate to the patients in case group and their percentages were calculated. The percentage of recovery rate was gradually increased between 6 hours-4 days of admission.

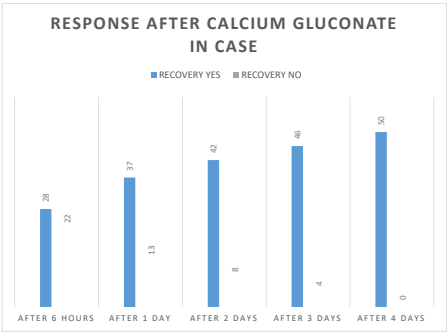

Fig. 1: Comparison of the Response After Giving Inj. Calcium Gluconate to the Patients of Case Group and their Percentage

Comparison between the response after calcium gluconate of the case and control group were observed. The higher number of complete recoveries was observed in <1 day of admission in case group as shown in (Fig. 2). The p-value is 0.001 <0.05 statistically significant were observed.

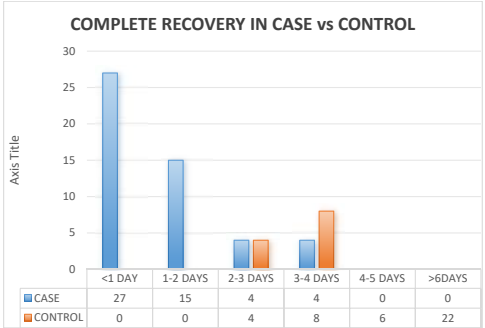

Fig. 2: Comparison Between the Response After Calcium Gluconate of the Case and Control Group

In this study, majority of the patients were aged between 41-50 years (24%), 22% of patients were aged between 31-40 years, 21% of patients were aged between 51-60 years of age, 16% of patients aged between 61-70 years, 135 of patients were aged between 21-30 years and 4% of patients were aged <20 years. Mean age in case group was 45.14±14.83 years and mean age in control group was 43.38±16.38 years. P value was 0.606, which was statistically insignificant. Response after giving Inj. Calcium gluconate in case group, after 6 hours were 28 patients, after 1 day were 37 patients, after 2 days in 42 patients, after 3 days in 46 patients and after 4 days in 50 patients. In case group complete recovery in <1 day was in 27 patients, in 1-2 days was in 15 patients, in 2-3 days in 4 patients, in 3-4 days in 4 patients. In control group complete recovery in 4 patients in 2-3 days, in 8 patients in 3-4 days, in 6 patients in 4-5 days and in 22 patients in >6 days. P value was 0.001, which was statistically significant.

CONCLUSION

Krait bites with neuroparalytic symptoms may be effectively treated with intravenous calcium gluconate therapy in combination with anti snake venom (ASV). Krait venom, also known as beta-Bungarotoxins, often induces presynaptic inhibition, where calcium functions as a neurotransmitter. We find that administering calcium gluconate infusion at an early stage in patients with Krait bite and neurotoxic envenomation results with reduced mortality and morbidity.

REFERENCES

1. Kularatne, S.A.M., 2002. Common krait (Bungarus caeruleus) bite in Anuradhapura, Sri Lanka: A prospective clinical study, 1996-98. Postgraduate Med. J., 78: 276-280.
2. Bawaskar, H.S. and P.H. Bawaskar, 2004. Envenoming by the Common Krait (Bungarus caeruleus) and asian Cobra (Naja naja): Clinical Manifestations and Their Management in a Rural Setting. Wilderness and Environ. Med., 15: 257-288.
3. Dixon, R.W. and J.B. Harris, 1999. Nerve Terminal Damage by ß-Bungarotoxin. The Am. J. Pathol., 154: 447-457.
4. Singh, G., H.S. Pannu, P.S. Chawla and S. Malhotra, 1999. Neuromuscular transmission failure due to common krait (Bungarus caeruleus) envenomation. Muscle and Nerve, 22: 1-10.0.
5. Lewis, R. and L. Gutmann, 2004. Snake Venoms and the Neuromuscular Junction. Seminars Neurol., 24: 175-179.
6. Anil, A., S. Singh, A. Bhalla, N. Sharma, R. Agarwal and I.D. Simpson, 2010. Role of neostigmine and polyvalent antivenom in Indian common krait (Bungarus caeruleus) bite. J. Infec. Public Health, 3: 83-87.

7. Pareek K.K., 2019. Medicines update. Management of snake bite., 2: 225-1120.
8. Abe T., A.R. Limbrick and R. Miledi., 1976. Acute muscle denervation induced by ß-bungarotoxin. Proc R Soc Lond B Biol Sci., 194: 545-543.
9. Chang C.C., T.F. Chen and C.Y. Lee., 1973. Studies of the presynaptic effect of ß-bungarotoxin on neuromuscular transmission. J Pharmacol Exp Ther., 184: 339-345.
10. Olek, A.J., 1980. Effects of a and ß bungarotoxin on motor neuron loss inXenopus larvae. Neuroscience, 5: 1557-1563.