ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at: www.ijmacr.com

Volume – 5, Issue – 1, January –February - 2022, Page No. : 290 – 294

A Study of Bleeding manifestations of Snake Bite in a rural population

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How to citation this article: Dr. Sourav Chattopadhyay, Dr. Srikanth Shetty, Dr. Aniket Sinha, Dr. Abhinav Chaudhary, Dr. Sumit Singh Kaushal, Dr. Suman Kumar Singh, "A Study of Bleeding manifestations of Snake Bite in a rural population", IJMACR- January – February - 2022, Vol – 5, Issue - 1, P. No. 290 – 294.

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Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background: Bleeding following snake bites by the can either is local or systemic. Bleeding at the site of the bite is due to the local action of the venom as a vasculotoxin. Systemic bleeding occurs with severe poisoning and appears to be mainly dependent on platelet deficiency and the co-existing defibrination syndrome appears to play a minor role in the initiation of bleeding.

Methods: A cross sectional observational study on 50 patients admitted with symptoms, signs and definitive evidence of snake bite. One healthy volunteer was taken who was matched with respect to age and sex with the case as a control. Cases of snakebite were analysed for the evaluation of coagulation disorder, after screening the patients with CT and BT even with normal value along with signs of systemic envenomation.

Results: Most common local reaction we observed was swelling at the site of bite. This had a variable extention in to the limbs concerned. Fang mark present in 87 patient. Bleeding from the site of bite were 73 cases. Ecchymosis was present near the bite in 5 patients.

Conclusion: The most common bleeding manifestation that we observed in our study was bleeding from the site of bite which was considered as evidence of systemic envenomation.

Keywords: Bleeding**,** CT and BT

Introduction

Bleeding following snake can either be local or systemic. Bleeding at the site of the bite is due to the local action of the venom as a vasculotoxin. Systemic bleeding occurs with severe poisoning and appears to be mainly dependent on platelet deficiency and the co-

existing defibrination syndrome appears to play a minor role in the initiation of bleeding. Thus in the clinical situation non-clotting blood with no overt bleeding can continue up to weeks when specific antivenene is not given.[1] Assessment of the severity of poisoning can easily be made at the bedside. Specific viper antivenene rapidly corrects the spontaneous bleeding and clotting defect of severe systemic poisoning but has no effect on local poisoning.[2]

Materials and methods

We conducted our cross-sectional study on patients admitted with symptoms, signs and definite evidence of snakebite. We considered following observation as definite evidences of snakebite. The presence of fang marks.

Inital laboratory evaluation by performing clotting time, bleeding time, at bedside. If they prolonged, we took it as evidence of envenomation with coagulation disorder. We also took evidence of envenomation by observing a local reaction confined to the site of bite with evidence of rapid extention of swelling and cellulities involving more than one joint. We took one healthy volunteer who was matched with respect to age and sex with the case as a control. We have analysed clinical features following snakebite even if the initial evaluation by CT and BT remained normal. This is because, from literature we gathered that the spectrum of venom toxicity following snakebite varies from haemototoxicity to neurotoxicity. Sometimes even combined manifestation of haematotoxicity and neurotoxicity is seen.

We assessed patients admitted in the emergency ward with evidences of snakebites by performing clinical examination as per the inclusion criteria. We considered all patients who came with a history of poisonous snakebite for the detailed clinical examination and evaluation of coagulation disorder. We compared results of all the tests with controls. We evaluated the results statistically later. We recorded the clinical features following snakebite in a prefixed proforma. These pre fixed proformas contained almost all the possible clinical feature in snakebite case. We evaluated these filled up proformas statistically later.

Results

Table 1: Hemoglobin Level

			Groups		Total
			Test	control	
HBC	<10	Count	15	14	29
		% Within Groups	15.0%	14.0%	14.5 %
	>10	Count	85	86	171
		% Within Groups	85.0%	86.0%	85.5%
Total		Count	100	100	200
		% Within Groups	100%	100%	100%

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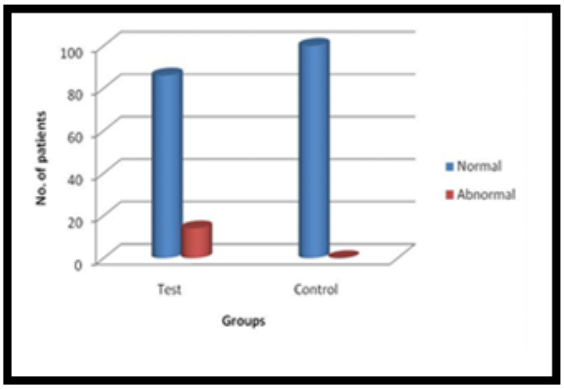
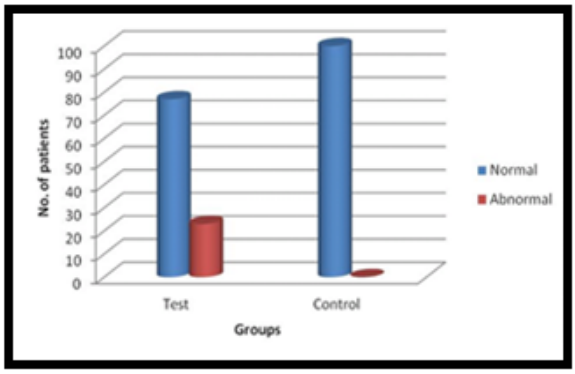
Table 2: Platelet Count

			Groups		Total
			Test	Control	
PLT	<1	Count	3	1	4
		% Within Groups	3.0%	1.0%	2.0%
	>1	Count	97	99	196
		% Within Groups	97.0%	99.0%	98.0%
Total		Count	100	100	200
		% Within Groups	100.0%	100.0 %	100.0%

Graph 1 : CT profile in cases and control



Graph 2: Prothrombin Time



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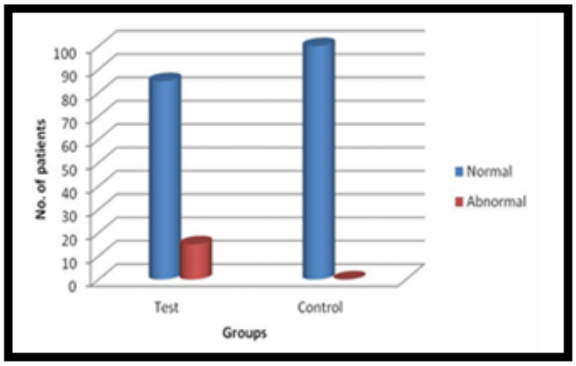
Graph 3: A PTT Profile



Discussion

The seasonal incidence in this study is similar to that of reported by Ahuja2 and Singh (1954) and Gupta et al (1960)3. The maximum incidence of snakebites in summer months in tropical and sub-tropical climates appear to be universal. In our study there was essentially no difference in the clinical picture of the patients bitten by Russel s Viper and of those bitten by Echis carinatus. This result is similar to that of the study reported by Bhat RN from Jammu in 19734.

The only effective and relevant treatment of snakebite poisoning to be advocated in a hospital, is the administration of anti-snake venom. As signs of systemic poisoning are not always clinically evident and as a patient with a nonclotting blood is potentially in danger of developing haemorrhagic syndrome, its not our practice to delay the administration of ASV as has been suggested by Reid5. We agree with Bhat s this conclusion in administering ASV. Immediately after admission when systemic poisoning is detected by finding defective coagulation ASV should be administered. Delay in the administration of ASV will delay the reversal of coagulation defect and is liable to endanger a patient s life by otherwise preventable haemorrhage.

In our study we observed that most of the patient (82%) came to hospital within six hour and received treatment and we observed very less complication compared to other studies with long duration between bite and admission to hospital. We strongly recommend to create public awareness regarding treatment of snakebite to reduce the complication.

Conclusion

The most common bleeding manifestation that we observed in our study is the bleeding from the site of bite. PT, APTT can remain normal or short in DIC 23 patients had laboratory evidence of DIC. (Prolonged PT, APTT) All coagulation defect can be reversed promptly with the administration of ASV. Delay in the administration of ASV will delay the reversal of coagulation defect and is more persistent will be the coagulation defect. The quantity of ASV required to reverse the coagulation defect will also be more. Blood transfusion helps to combat bleeding manifestation by providing clotting factor.

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