Case Report
Toxicology International, *May-August 2017 / Vol-24 / Issue-2, 211-215*
ISSN (Print) : 0971-6580
ISSN (Online) : 0976-5131
DOI: 10.22506/ti/2017/v24/i2/162427

Myocarditis Complicating Viper Snake Bite in a Child

K. Jagadish Kumar*, Srujan, Ashok Chowdary, Ravi Kumar, Keerthi Gupta, Manjunath VG

Department of Pediatrics JSS Medical College, JSS University, Mysore.
vghunsur@rediffmail.com

Abstract

Snake bites are important medical emergencies in clinical practice. Viper bite envenomation classically comprise of local cellulitis, coagulopathy and nephrotoxicity. Cardiovascular manifestations are rarely reported which usually include non-specific ECG changes.We report 12 year old boy who developed shock and myocarditis following viper envenomation along with coagulopathy and kidney injury.

Keywords: Viper envenomation, Shock, Myocarditis, coagulopathy, AKI

Received: 10-09-2016 **Revised:** 14-03-2017 **Accepted:** 18-03-2017

Introduction

Snake-bites are one of the important well-known medical emergencies in the world[1].In the Asia-Pacific region there was an estimate of 237379–1184550 snake envenomings with 15385–57636 deaths in 2008[1].Snake-bite was included in the WHO list of neglected tropical diseases in 2009[1]. In India as high as 50000 deaths occur each year due to snake bites[1].Viper bite envenomation classically characterized by a local tissue reaction, bleeding manifestations and nephrotoxicity; Cardiotoxicity is a rare manifestation of viper bite[2,3].The cardiovascular manifestations include ECG abnormalities,arrthymias,myocardial ischemia and infarction[2,3,4,5,6].Myocarditis have been reported in animals following viper envenomation[7]. However myocarditis due to viper envenomation in humans is rare[7].Herewith we report 12 year old boy who developed AKI,coagulation abnormalities,shock and myocarditis following viper envenomation.

Case report

A 12 year old boy was brought with viper snake bite on right leg at 8.30PM to pediatric ward at 10.30PM.After the bite he was taken to local quack where multiple incisions were made to squeeze the venom.There was no history suggestive of nervous system involvement or bleeding tendencies.On examination his vitals were normal with temperature 98.6^{0}F,RR20/min,PR 68/min,BP of 114/70mm of Hg,CFT< 3seconds and oxygen saturation of 100% at room air.Systemic examination was unremarkable.Local bite examination revealed multiple incision marks with tendeness at the bite mark on right leg 3 cm above ankle along with swelling upto middle leg.In view of local cellulitis 10 vials of Anti Snake Venoum(ASV) was started along with inj Ampicillin+cloxacillin and metronidazole.After 6 hours of ASV his PT/INR ratio and aPTT was abnormal,therefore another 5 vials of ASV added.Again after 6 hours PT/INR ratio and aPTT

* *Author for correspondence*

was prolonged therefore another 5 vials of ASV given. Investigations and the following sequence of events are depicted in the Table 1 and flow chart 1.Overall 35 vials of ASV given over 2 days in view of shock,AKI,coagulation abnormalities and myocarditis.By 8th day his vitals were stable without inotropes and diuretics.After 14 days of admission Echocardiography showed ejection fraction of 48%,he was discharged.During the follow up after 10 days Echocardiography showed ejection fraction of 54% with good left ventricular function.

Discussion

Viper envenomation mainly characterized by a local tissue reaction, hemorrhagic manifestations and nephrotoxicity[2,3]. Coagulopathy and acute renal failure are the cause of complications that lead to death[8]. Cardiovascular manifestations are rarely reported which include ECG abnormalities,arrthymias,myocardial ischemia and infarction[2-6].Out of 336 victims of Russell's viper snake bites,envenomation manifested in 92% of patients.The manifestations are local swelling 92%, coagulopathy 77%, neurotoxicity 78%, nephrotoxicity 18% and cardiac effects 3-12%.Cardiac effects were in terms of ECG abnormalities like tall T waves(12%) and T wave inversion(3%) of patients. One child developed typical chest pain,elevated cardiac enzymes and ECG revealed a non-Q wave myocardial infarction[5]. Out of 55 proven Russell's viper bite and 154 suspected viper bite cases, they observed local envenoming(91.9%),coagulopathy(76%), renal dysfunction (18.6%) and neuromuscular paralysis(69.9%).Only 5.7% had cardiovascular dysfunction., but the details are not reported[8].

Our child developed profound hypotension after 18 hours of snake bite inspite of giving 20 vials of ASV.Snake bite can results in profound hypotension and shock[1]. The causes for shock include vasodilatation,cardiotoxicity[cardiovascular effects of the venom], hypovolaemia, haemorrhage, septicaemia, antivenom reaction or rarely primary anaphylaxis induced by the venom itself[1].Inspite of coagulation abnormalities he never had bleeding tendencies.Therefore probably shock may not be due to hypovolemia or haemorrahage.He received ASV without any anaphylaxis or reaction.Eventhough local bite mark swelling was progressive,septicemia is unlikely as the child developed shock within 24 hours.However we started antibiotics to cover sepsis also along with inotropes.Most probably shock may be due to cardiovascular effects of the venom causing myocarditis as supported by increase in the levels of CPK-MB,ECG and Echocardiography findings.

In snake envenomation cardiac complications usually include non-specific ECG changes only[4].ECG of our child had 3mm ST-T segment depression suggestive of myocardial ischemia.In a Srilankan study out of 45 viper bite patients only 2 patients had transient ECG changes[6]. In myocarditis ECG is usually abnormal and the changes are neither specific nor sensitive[9].ECG changes described in snake bite include sinus tachycardia,sinus arrhythmia, sinus bradycardia, atrioventricular rhythm,non-specific T wave inversion,tall T-wave, heart block, myocardial ischemic and infarction changes[2-6].

A study from Jammu & Kashmir showed significant statistical elevation in CPK-MB in 24 hours and 72 hours of the snake bite in haemotoxic group when compared to neurotoxic and nonenvenomated groups[10].In our child also there was elevation of CPK-MB by 24 hours. However out of 45 victims of viper bite, troponin T levels were not elevated in any of them in a study from Srilanka and authors concluded that myocardial damage did not seem to be an important feature of viper bite[6].

Echocardiography revealed severe left ventricular global hypokinesia with left ventricular ejection fraction of 25%.The gold standard for diagnosis of myocarditis is an endomyocardial biopsy, which is an invasive procedure[9]. Considering the clinical presentation increased levels of CPK-MB,ECG and Echocardiography findings we entertained the diagnosis of myocarditis. Shock in our child responded to ASV and inotropes. Other authors have also used dopamine to treat snake bite shock[1,5]. Our child underwent frequent Echocardiography once in 3 days to asses left ventricular function and we continued dobutamine till left ventricular ejection fraction increased to 45%.After discharge follow up Echocardiography revealed improvement in the ejection fraction to 54%. Myocarditis have been reported in animals following viper envenomation[7]. However myocarditis is a rare complication., Bhat et al reported myocarditis along with cerebrovascular complications following viper bite[7]. Myocarditis is postulated to occur either due to direct effects of the toxin on the myocardium or hypersensitivity to the venom [7].

Table 1. Investigation chart following viper bite.

	Platelets [/cumm]	Urea [mg/dl]	Creatinine[mg/dl]	PTsec/INR ratio	aPTTsec/ ratio	CPK-MB [ng/ml]	ABG P^H pCO_2 [mmHg] pO_2 [mmHg] HCO_3 [mmol/L]	AST [U/L]	ALT [U/L]	Echocardiography
Admission	2.24Lak	20	0.9	18/1.4	27/0.83	4.3				
Day 1	67000	44	1.7	27.6/2.25	31.2/0.95	12.81	7.37,	64	50	
6 Hours		52	1.7	27.7/2.26	31/0.95		28.6,			
12 Hours		57	1.9	29/3.02	34/1.03		91.5,			
20 hours							16.3			
Day 2	35000	72	1.5				7.31	95	32	LVEDD= 43
12 Hours				30.5/2.54	33.9/1.03		29,			mm IVSD= 13
24 Hours				35.1/3.02	34/1.03		86.7			mm, Severe
							16.4			left ventricular
										global hypo-
										kinesia with
										left ventricular
										ejection fraction
										of 25%
Day 3	49000	44	1	24.8/1.97	36.2/1.1			75	22	
Day 4	73000	26	0.7				7.4			
							35.6			
							65.2			
							19.3			
Day 5										Severe left ventricular global hypokinesia with left ventricular ejection fraction of 34%
Day 6	85000	18	0.7	15.3/1.1	29.2/0.8					
Day 8	1.74Lak									left ventricular ejection fraction of 45%

Echocardiograpy on 2nd day:- Normal Left Ventricular End Diastolic Dimension (LVEDD, 43 mm); normal Inter Ventricular Septal thickness in Diastole (IVSD, 13 mm);severe left ventricular global hypokinesia with left ventricular ejection fraction of 25% .There was no evidence of valvular abnormalities or pulmonary hypertension.

Left ventricular ejection fraction of 48% on 11th day, Discharge follow up after 10 days showed ejection fraction of 54%.

Ultrasonography:-Both kidneys enlarged,grade 3 medico renal disease and minimal acitis on 2nd day.

d-Diamer=0.83[normal upto 0.5 microgm FEU/ml] on 3rd day, Blood Culture sterile.

Figure 1. Sequence of events after viper bite.

After 18 hours of admission	Developed hypotension,BP 74/56 mm of Hg,CFT>5sec,PR136/min,feeble RR24/min, $Spo_2$98%, gallop rhythm present.Started on normal saline bolus 20ml/kg given 3 boluses, dopamine 10microgram/kg/min &O_2started. No response therefore increased dopamine 15microgram/kg/min, BP still low[70/54 mm of Hg]warm peripheries. Started on noradrenaline0.5 mic gram/kg/min and antibiotics injection pipericillin+tazobactum,linezolid &metranidazole.
18 hours to 24 hours of admission	PR144/m ,BP maintaining above 50th centile but developed tachypnoea RR 48/min,gallop rhythm present,Spo_2 95%,CFT<3sec,chest x-ray revealed pulmonary odema/ARDS picture. ABG=P^{H}7.37,$pCO_2$28.6, pO_2 91.5, HCO_3 16.3.Therefore inj Furesamide 1mg/kg given.
25 hours to 48 hours of admission[2nd day]	RR 38/min,$Spo_2$98%,BP 112/70 of Hg,PR 138/min, peripheries cold[on dopamine and noradrenaline],CFT>3sec,gallop rhythm present.Echocardiography revealed ejection fraction of 25 %,severe global hypokinesia of left ventricle. Considering the clinical presentation with gallop rhythm & shock,increasing levels of CPK-MB,ECG changes, ECHO findings we entertained the diagnosis of myocarditis.Stopped noradrenaline and started on epinephrine 0.5 micgram/kg/min with dobutamine10microgram/kg/min .
	For 24 hours BP was normal,RR30/min,PR120/min,$Spo_2$98%,CFT<3sec
49 hours to 72 hours of admission [3rd day]	PR 124/min,RR 28/min,Spo_2 96%, BP 96/60mm of Hg, CFT<3sec,Warm peripheries. Started tapering dopamine till 5micgram/kg/min.Continued epinephrine & dobutamine.
4th day	PR110/min,RR40/min, BP 96/60mm of Hg, CFT<3sec, 3 cm hepatomegaly , basal crepitations present .Chest x-ray pulmonary odema.Inj Furesamide infusion 0.5 mg/kg/hour started and dopamine stoped
5th day	RR 38/min,PR118/min, peripheries warm ,BP 94/60 of Hg, Spo_2 98 %, no gallop,CFT<3sec. Ejection fraction of 34 %.Epinephrine tapered over 24 hours. Dobutamine 10microgram/kg/min and Furesamide infusion continued.
6th day	Taking orally,RR 32/min,PR 106/min, $Spo_2$100%,CFT<3sec, BP 96/50 of Hg, no gallop, 2cm hepatomegaly,no crepitations.Started tapering Furesamide infusion.,stopped after 8 hours. Dobutamine continued.
7th day	Taking orally,RR 30/min, PR 122/min, BP 98/64 of Hg, Spo_2 100%, CFT <3sec. no gallop rhythm. Started tapering dobutamine over 24 hours.
8th day	Taking orally,PR 100/min,RR 28/min, BP 94/54 of Hg,Systemic examination unremarkable. Ejection fraction of 45 %.Not on inotrophes/Furesamide.

Our child also had thrombocytopenia,coagulation abnormalities and AKI. All abnormalities returned to normal with 35 vials of ASV. Thewjitcharoen and Poopitaya opined that the clinician should be prepared to manage a rare manifestations of snakebite[3].Our child is a typical example for this statement.

To conclude in snake bite envomation we should anticipate the complications,need close clinical and investigative monitoring. Even though coagulopathy and AKI are the common complications, myocarditis is also most important rare complication of viper envomation. High index of suscpicion should be kept when there is shock following viper snake bite.

Conflict of Interest

None Declared

References

1. David A Warrell. Guidelines for the management of snake-bites. World Health Organization, New Delhi. 2010. India.
2. Agarwal A, Kumar T, Ravindranath K S, Bhat P, Manjunath C N, Agarwal N. Sinus node dysfunction complicating viper bite. Asian Cardiovascular and Thoracic Annals. 2015; 23: 212-214.
3. Thewjitcharoen Y, Poopitaya S. Ventricular Tachycardia, A Rare Manifestation of Russell's Viper Bite: Case Report. J Med Assoc Thai 2005; 88:1931-1933.
4. Moore R S. Second-degree heart block associated with envenomation by Vipera berus Archives of Emergency Medicine.1988;5:116-118.
5. Kularatne S A M. Epidemiology and clinical picture of the russell's viper (daboia russelii russelii) bite in Anuradhapura, Sri lanka: a prospective study of 336 patients. Southeast Asian J Trop Med Public Health.2003;34:855-861.
6. Seneviratne S L, Gunatilake S B, Fonseka M M, Adhikari A A, de Silva H J. Lack of myocardial damage following Sri Lankan Russell's viper and hump-nosed viper bites. Ceylon Med J 1999;44:70-73.
7. Bhatt A, Menon A A, Bhat R, Ramamoorthi K. Myocarditis along with acute ischaemic cerebellar, pontine and lacunar infarction following viper bite. BMJ Case Rep. 2013; 2013: bcr2013200336.
8. Kularatne S A M, Silva A, Weerakoon K, Maduwage K, Walathara C, Ran Paranagama R, Mendis S. Revisiting Russell's Viper (Daboia russelii) Bite in Sri Lanka: Is Abdominal Pain an Early Feature of Systemic Envenoming?. PLoS One. 2014; 9(2): e90198.
9. Caforio A L P, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix S B et al.
10. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2013;34:2636-2648.
11. Gupta P, Mahajan N, Gupta R, Gupta P, Chowdhary I, Singh P, Gupta A K, Kakkar M. Cardiotoxicity Profile of Snake Bite. JK Science.2013;15:169-173.