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CASE REPORT

VARYING A V BLOCK COMPLICATING SNAKE BITE - A CASE REPORT

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ABSTRACT: Snake venom toxicities comprise mainly bleeding disorders and nephrotoxicity. Cardiotoxicity is a rare manifestation of snake bite. We describe the case of a previously healthy 23-year-old man who developed coagulopathy and AV node dysfunction following snake bite. Electrocardiography showed all variations of AV conduction dysfunction. This is the first account of AV node dysfunction caused by a snake bite with cardiotoxicity presenting as atrioventricular block.
KEYWORDS: Sick sinus syndrome, snake bites, AV conduction blocks, neurotoxicity, AV dissociation.

INTRODUCTION: Snake bite is a common problem in rural areas of Asia, and the cause of several thousand deaths each year. Snake bite is usually characterized by a local tissue reaction, hemorrhagic manifestations, and nephrotoxicity; cardiac involvement is rare. There are number of case reports regarding myocarditis and myocardial infarction[1] following snake bite but not many cases have been described with the involvement of the conducting system of the heart. Here we present a case with AV conduction abnormality following snake bite.

Case report A 23 year old male presented to emergency ward 24 hours after snake bite with history of blurring of vision, diplopia and decreased urine output. On admission his pulse was 54 beats/min, irregularly irregular. BP-120/70 mm of Hg, RR-14 breaths/min. On local examination fang mark was present over left 2nd toe with no signs of local reaction and systemic manifestation of envenomation. Investigations done on the same day revealed ECG with normal rhythm(fig 1), troponin T – negative, serum electrolytes were normal, WBC count- 26,700 cells/mm^3, blood urea- 62 mg/dl, S.creatinine- 2.5 mg/dl, platelet count- 50,000 cells/mm^3, WBCT> 20 mins (table 2). The patient was immediately started on IV fluids, ASV, antibiotics and analgesics. Inspite of giving ASV 10 hours later he complained of worsening of diplopia and patient developed tachypnea, cyanosis, ptosis, muscle weakness, fasciculations all suggestive of neuromuscular manifestations of envenomation. He was shifted to MICU and was intubated.

Repeat ECG (after 6 hrs.) showed 1st degree AV block (fig 2). 24 hours after admission patient developed hypotension and was started on inotropic support and steroid. ECG showed tachycardia, non-specific ST, T wave changes with AV dissociation (fig 3).On the 2nd day he was hemodynamically stable and inotropic support was discontinued. His neuromuscular weakness also improved and was extubated. ECG showed 1st degree AV block with persistence of non-specific ST, T wave changes (fig 4). ECG repeated on 4th-11th day showed persistent 2nd degree AV block and he was hemodynamically stable. In view of persistent bradycardia he was started on isoprenaline on 6th day, by 10th day heart rate improved to 93 beats/min. On the 12th day ECG was normal. Patient was discharged on the 15th day with HR of 84 beats/ min. On the 1st follow up after 1 week ECG was normal with HR of 84 beats/ min(fig 5). ECG changes from day of admission till discharge has been described in table 1.

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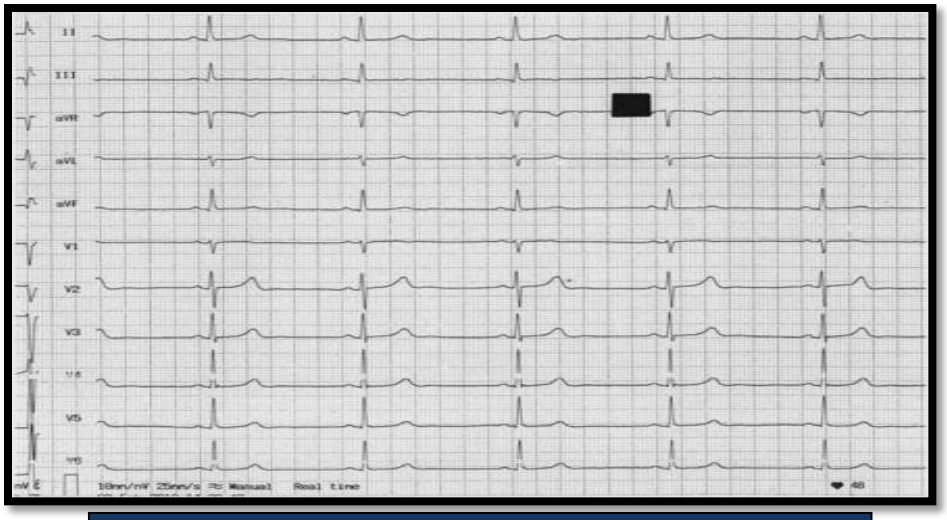

Fig. 1: (at time of admission normal rate and rhythm)

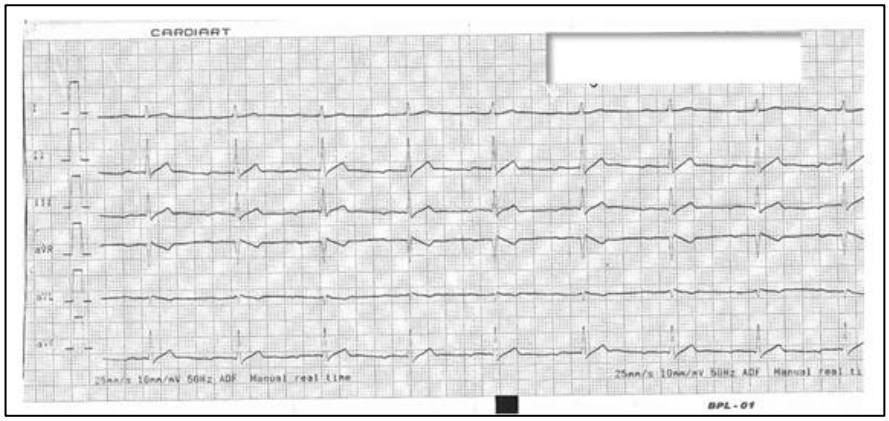

Fig. 2: (Day 1- 10 hrs after admission showing 1st degree AV block)

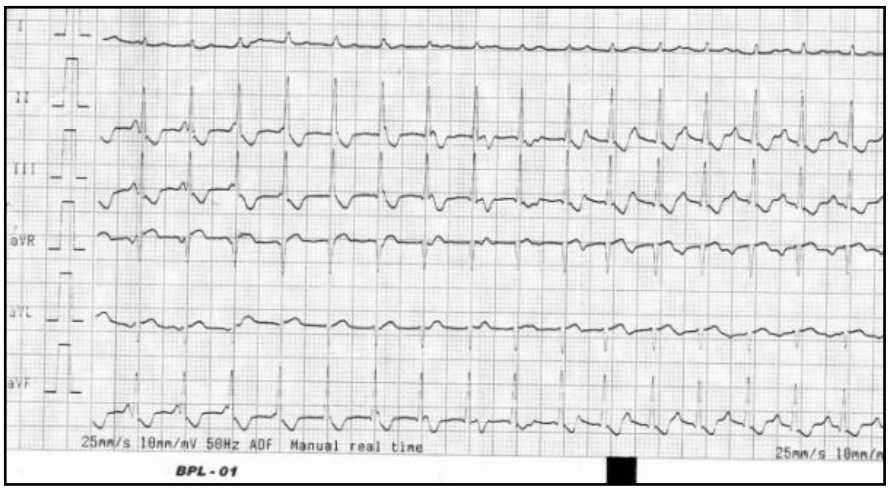

Fig. 3: On Day 2 showing AV dissociation

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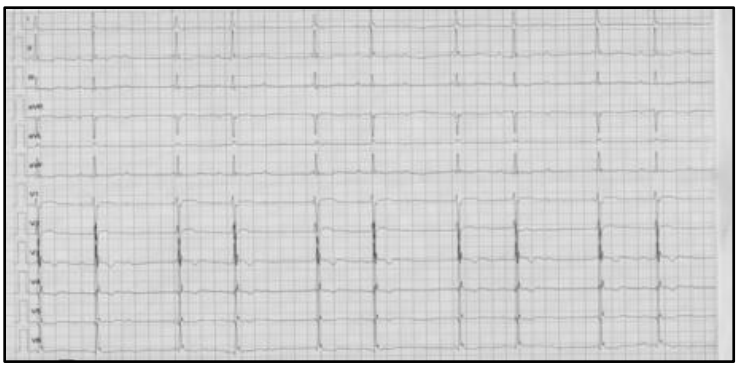

Fig. 4: (Day 5 showing 2nd degree AV block- Mobitz type I)

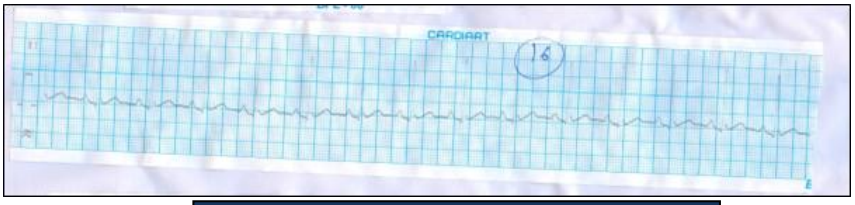

Fig. 5: Day 14 showing normal ECG

Day	HR/min	PR interval	ST-T wave changes	Conclusion
On admission	50	Normal	No changes	normal
6 hrs	40	Varying	ST ↑ V2	Varying 2nd degree block type I
10 hrs	65	0.28 sec	ST ↑ V1,V2,T inversion V1, V2,V3	1st degree AV block
24 hrs	150	Progressive shortening	ST ↓ & T inversion V1, V2,V3	AV dissociation,? Myocarditis
48 hrs	107	0.24 sec	ST ↑ V1,V2, ST ↓ & T inversion II,III,avF,V3-V6	1st degree AV block
4th day	53	0.12 sec	ST coving V2, T inversion V3,V4	? 2nd degree AV block 2:1
5th day	50	Varying	ST ↑ V1,V2, T inversion II,III,avF,V3-V6	2nd degree AV block type 1,3:2
8th day	50	Varying	T inversion II,III, avF,V1-V6	Varying 2nd degree block 3:2,2:1,4:3
9th day	60	Varying	ST ↑ V2, T inversion II,III, avF,V3-V6	Varying 2nd degree block 3:2,2:1
10th day	93	Varying	ST ↑V1, T inversion V2-V4	Varying 2nd degree block 5:4,4:3
11th day	60	Varying	ST ↑V2, T inversion V1,V2,V3	2nd degree AV block type 1 4:3
12th day	90	0.16 sec	ST ↑V2, T inversion V1,V2,V3	Normal
14th day	84	0.16 sec	Normal	Normal

Table 1: ECG changes from day of admission to discharge

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Day	Total Count – cells/mm^3	Blood urea mg/dl	S.creatinine mg/dl	S.potassium-meq/L	Platelet count-cells/mm^3	WBCT-min
1	26700	62	2.5	4.49	50000	>20
2	28100	65	1.3	4.8	70000	>20
3	31700	40	0.8	4.43	141000	<20
4	27800	42	0.8	3.57	180000	<20
12	6400	39	0.8	3.84	190000	<20

Table 2: Hematological & biochemical findings

DISCUSSION: Cardiac complications and ECG abnormalities following snake bite has been reported number of times but not many case reports with involvement of conduction system of the heart has been described. In this case species of snake has not been identified and troponin T was negative. Varying AV block was observed from day1 to day 11 and ECG became normal on day 12.

ECG abnormalities or involvement of the myocardium has been observed following envenomation by a no of different species of snakes including viper berus, atractaspis engaddensis, Echis ocellatus and Calloselasma rhodostoma.

Explanation for ECG changes in our case might be because of various mechanisms. In a prospective study conducted by David G et al[2] in Port Mores General hospital in PapuaNew Guinea between march 1990 and june1992 they studied ECG's of 139 patients envenomated by Elapid snakes in that 76 patints had ECG abnormalities,commonest abnormalities were septal T wave invertion and bradycardia including AV block. Bradycardia/1^0 AV block were seen in 2 of the taipan envenomation and 2^0 AV blockin one case bitten by death adder. ECG abnormalities are most likely to have been caused by a direct toxic effect of a venom component either on the heart or on the innervation of the heart.In Taipan bites taicatoxin a CCB might be responsible for the ECG abnormality.[2]

In a study done by Nayak KC et al in SP Medical College,Bikaner they studied 30 cases of snake bite cardiotoxicity was seen in 30% in that AV block in 3.3% only.[3]

Tony JC et al reported a case of acute MI complicating snake bite in a 47 year old man.It was presumed to be due to sarafotoxin in snake venom having vascular toxic effect leading to vasospasm in coronary vessels and also delay AVconduction.[4]

Various other mechanisms have been suggested –directly acting cardiotoxin[5], myotoxin causing cardiac muscle damage,[6] coronary vasospasm,[7] electrolyte disturbance,[8] severe hypotension[9] and disturbance of autonomic innervation of the heart.

CONCLUSION: This case report describes the fatal outcome of snake bite in a 23 yr. old male due to cardiotoxic manifestation of snake bite. Toxic myocarditis seems unlikely to be the cause of the ECG changes because cardiac enzymes were not elevated and there were no findings suggestive of myocarditis on ECG or echocardiography. One should consider a direct toxic effect of venom on the sinus node. It is believed that snake venom modifies the electrophysiological properties of the cardiac cell membrane, and can have a profound effect on impulse generation and conduction. However, the exact pathophysiology is not known. All physicians should be prepared for prompt treatment of even such a rare manifestation of snake bite. This case also suggests that victims may need prolonged

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cardiac monitoring even when the acute phase is over. Studies are required to elucidate the effect of snake venom on the electrophysiological properties of the cardiac cell membrane.

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