CARDIAC INVOLVEMENT IN SNAKE BITE

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Introduction

Cardiac complications are not prominent features of snake bite and the clinical picture is usually dominated by neurological, haematological and vascular damage by the snake bite toxins. Myocardial involvement is seen on occasions and may rarely contribute to morbidity and mortality. T wave abnormalities are the most common manifestation of myocardial involvement, although ST segment depression, QRS prolongation and AV conduction defects may also be seen rarely. ECG changes are usually transient but when persistent they are attributed to direct myocardial damage due to the toxin [1]. One such case who showed these rare ECG changes following snake bite is reported.

Case Report

61 year old female patient, not a known case of ischaemic heart disease, hypertension, diabetes mellitus or any other illness was hospitalised with the history of snake bite over the left foot. She had tingling sensation and numbness over the limb up to knee joint associated with progressive swelling upto mid calf region in next 6 hours. Clinical examination revealed an elderly lady, afebrile, having irregularly irregular pulse of 148/min and BP 110/70 mm Hg. Systemic examination was not contributory. Investigation showed abnormal coagulation parameters (BT-8'20" CT-25'), platelet count 1,80,000/cmm, Hb 11gm/dl, TLC 7600/cmm, blood urea 30 mg/dl, serum creatinine 0.6 mg/dl, cardiac enzymes as available were SGOT 30 IU/l and SGPT 18 IU/l. Serial estimation over the next 3 days did not show any abnormalities in enzyme levels. Other investigations like serum electrolytes, serum calcium, prothrombin time and X-ray chest were within normal limits. Fi-

Fig. 1 : Cardiac involvement in snake bite

brinogen and fibrinogen degradation product estimation could not be done. ECG showed an irregular rhythm, junctional ectopics, RBBB and ST depression of 1-4 mm (Fig-1). Patient was managed with anti-snake venom, xylocard and supportive measures. Pulse rate returned to normal in next 48 hours while coagulation parameters reverted to normalcy in 96 hours. Patient made an uneventful recovery. Serial ECG showed reversal to sinus rhythm in 48 hours.

Discussion

Snake venom cannot be regarded as a single toxin. The variation of venom composition from species to species explains the clinical diversity of snake bite. There is also considerable variation in the relative proportion of different venom constituents within a single species within a geographical distribution at different seasons [2]. In patients bitten by snake, the signs and symptoms are explained by fear, direct action of the various venom components on tissues, indirect effects such as complement activation and auto pharmacological release of endogenous vasoactive substances. The neurotoxins and cardiotoxins of Elapid and Hydrophid venom are of small molecular size and carry strong positive charge, which is reflected in their low antigenicity and rapid lethal absorption. The lethal potency of cobra venom cardiotoxin is 1/20 of its neurotoxin. The primary action of cardiotoxin is directly on cell membrane, causing many effects on the skeletal, cardiac, smooth muscles, nerves and neuromuscular junctions, thus contributing to circulatory and respiratory paralysis and cardiac asystole [3]. The pharmacological action of cardiotoxin has been shown to be due to an irreversible depolarisation of the cell membrane transport mechanism and asystolic cardiac arrest possibly due to release of Ca^{++} from the surface membrane of the myocardium [4].

Many types of snakes are attributed to be causing cardiovascular symptoms and ECG changes. Patients envenomed by Burmese Russell's viper may bleed into anterior pituitary gland (Sheehan's Syndrome). Hypotension and shock are common in patients bitten by North American Rattle snakes, Bothrops, Daboia and Viper species. Direct myocardial involvement is

suggested by an abnormal ECG in cardiac arrhythmia as noticed in this case also. In European viper bite, ECG changes include flattening or inversion of T wave, ST elevation, second degree heart block, brady or tachyarrhythmias, atrial fibrillation and myocardial infarction [4].

Myocardial infarction and circulatory collapse may be due to DIC and/or direct myocardial toxicity. ECG abnormalities have been reported as unusual but important observations. Sinus bradycardia, ST-T changes, various degrees of AV block and evidence of hyperkalemia have been described, especially in envenomation by Viperidae, Australian Elapids and Atractaspidae [2]. Similar changes have been seen in this case also which was of viperine bite. However there was no evidence of hyperkalemia electrocardiographically or by serum estimation. Transient ECG changes of myocardial infarction have also occurred following adder bite, probably due to direct toxic effects of snake venom on myocardium. ECG changes present in this case were also transient in nature, indicating that there was no myocardial damage. This is also seen by normal cardiac enzyme levels. Adder bites have resulted in death from circulatory collapse and myocardial infarction secondary to severe hypo-

tension. Milder cases having transient angina pectoris and reversible T wave abnormalities have been described due to Malayan viper, where DIC was the postulated mechanism. Death may occur from circulatory collapse, myocardial depression or myocardial infarction due to hypotension, coronary artery thrombosis or vasospasm [6].

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