Case Report

Ventricular Tachycardia, A Rare Manifestation of Russell's Viper Bite: Case Report

Yotsapon Thewjitcharoen MD*,
Supaporn Poopitaya MD*,

** Chaophya Abhaibhubejhr Hospital, Prachinburi*

Snakebite is a common problem in rural areas of Asia. Russell's Viper toxicities are mainly bleeding disorder and nephrotoxicity. Cardiotoxicity is a rare manifestation of Russell's Viper's bite. A healthy man presenting with a Russell's viper bite developed cardiac arrest from ventricular tachycardia. He was successfully treated by cardiovertion and amiodarone. Subsequently, antivenom for Russell's Viper was administered for correction of a bleeding disorder.

Keywords: *Ventricular tachycardia, Arrhythmia, Russell's viper bite*

J Med Assoc Thai 2005; 88 (12): 1931-3
Full text. e-Journal: *http://www.medassocthai.org/journal*

A 46-year-old Thai man who lived in an urban area of Prachinburi province presented with a snakebite at his left ankle he had received while he was walking in his farm. He visually confirmed that he was bitten by a Russell's Viper. He had been in good health. He had no history of taking any medication. In the emergency room, he remained healthy and his vital signs were stable. Venous clotting time was 9 minutes. There was a small fang mark over his left ankle. About two hours after he was bitten, he suddenly developed cardiac arrest. His blood pressure couldn't be measured. His EKG monitor showed monomorphic ventricular tachycardia. He was intubated and cardioverted with an electrical current of 150 Joule, after which his rhythm converted to sinus rhythm over a short span of time before converting back to ventricular tachycardia. Amiodarone was administrated in order to terminate the ventricular tachycardia. He was admitted to the intensive care unit and was treated by continuous intravenous drip of amiodarone. Laboratory chemistry revealed normal renal function and electrolytes. A coagulogram showed an activated partial thromboplastin time of more than 150 sec (normal control at 34 sec) and prothombin time of more than 100 sec (INR > 13). Cardiac enzymes were obtained after he was cardioverted. Creatine phosphokinase and CK-MB were 416 U/L and 38 U/L respectively. Six vials of Russell viper's antivenom and four units of Fresh Frozen Plasma were administered in order to correct the coagulopathy.

On the second day of admission, ventricular tachycardia occurred again and the amiodarone titration was increased, but the patient remained unconscious. Venous clotting time was acquired every 6 hours. Repeated doses of antivenom were administered. Cardiac enzymes were again checked 6 hours later, with creatine phosphokinase 13,000 U/L and CK-MB 931 U/L. Serum creatinine was 1.5 mg/dl. Rhabdomyolysis was diagnosed and the patient was treated by vigorous intravenous fluid replacement to prevent renal failure. He was monitored closely and received supportive treatment in the intensive care unit for 7 days, then he finally regained consciousness. Intravenous amiodarone was switched to oral amiodarone 200 mg/day for 7 days. A total of 12 vials of Russell's viper antivenom had been administered to correct the coagulopathy. There was a prolonged QT interval (QTc 0.55 sec) on the ECG before the patient was discharged. He completely recovered and was discharged from the hospital. Unfortunately, the patient was lost to follow up after he was discharged. Without the repeated ECG and the patient's clinical record after being discharged, the long term result of venom to the heart is not fully understood.

Correspondence to : Thewjitcharoen Y, Chaophya Abhaibhubejhr Hospital, 32/7 Moo 12 Prachianusorn Rd, Ampur. Muang, Prachinburi 25000, Thailand. Phone: 0-3721-1523, E-mail: kamijoa@hotmail.com

Discussion

Snakebite is still a common problem in rural areas of many tropical countries, including Thailand. The estimated annual mortality rates due to snakebite in Pakistan and India are 1.9 and 5.4/100,000 population respectively[1]. Venomous snakes are classified by their system of toxicity, however the poison of some venomous snakes works in more than one way, such as the Russell's viper in Taiwan which has both neurotoxic and hematoxic properties[2].

Russell's viper (*Daboia russelii*) is a venomous snake which has a special sense organ, the pit organ, to detect warm-blooded prey. In Thailand, they are found mainly in the farms around the central part of Thailand[3]. The bite of a Russell's viper normally leads to nephrotoxicity and coagulopathy, but diverse clinical manifestations also occur. In a large series of Russell's viper bite cases from Sri Lanka, envenoming manifested in 310 (92%) patients as follows: local swelling 92%, local necrosis 8.9%, coagulopathy 77%, neurotoxicity 78%, nephrotoxicity 18%, cardiac effects 3-12% and myotoxicity 14%[4].

Snake venoms contain more than 20 different constituents, mainly proteins, including enzymes and polypeptide toxins. Russell's viper venom contains several different procoagulants which activate different steps of the clotting cascade. Phospholipase A2 is the most widespread venom enzyme which can be found in almost any venomous snake. It contributes to myotoxicity, neurotoxicity, cardiotoxicity, and hemotoxicity[5]. Cardiovascular manifestations are rarely reported in the literature, but one review from India notes tachycardia, hypotension, EKG changes, acute myocardial infarction and cardiac standstill from hyperkalemia[6]. Russell vipers from other countries such as Burma, India and Sri Lanka may have a higher severity of toxicity than in Thailand.

In the authors situation, the presented patient suddenly developed cardiac arrest from ventricular tachycardia, about two hours after he had been bitten by a Russell's viper. The authors believe that the Russell viper's toxin was responsible for the ventricular tachycardia, based on the patient's history and successful treatment with antivenom and an antiarrhythmic agent. On reviewing the literature, the authors found only one report from Pakistan describing fatal cardiac arrhythmia from snakebite[7], but unfortunately in this case the specific venomous snake was not reported. The pathogenesis of ventricular tachycardia from snake bite is not well understood. It is believed that snake venom modifies the electrophysiological properties of the cardiac cell membrane and can have profound effects on impulse generation and conduction throughout the heart. There was a prolonged QT interval on the ECG before the patient was discharged. This could be explained by the authors' belief that the cardiac cell membrane damage resulted from the venom is permanent although the venom effect is thought to have disappeared in a short period of time.

To the best of the authors' knowledge, this patient is the first case report of cardiac arrhythmia from Russell's viper bite in Thailand, and also the first case report in the world of a Russell's viper bite victim who subsequently suffered cardiac arrhythmia but, after treatment, survived without any complications.

It is also of interest to note that in the presented case, ventricular tachycardia occurred in the second day following admission, which indicates the residual presence of venom in the bloodstream. In such cases, repeated antivenom administration is indicated to neutralize any remaining venom.

Conclusion

The authors present the case of a rare manifestation of Russell's viper bite. Physicians should always be aware of the potential for unusual complications, even in seemingly ordinary cases. Everyone knows that "common things occur commonly," but all physicians in settings where there are venomous reptiles should be prepared for prompt treatment of rare manifestations of snakebite. All snakebite victims require close monitoring of the patient and supportive treatment must be continued until the compromised organs and tissues have had time to recover.

Acknowledgements

The authors wish to thank Thepsoparn M, MD and Mr Dave Patterson for their helpful review of the manuscript and Mr Montree Cheawbumroongkeat from Queen Saovabha Memorial Institute, Thailand for his excellent advice.

References

1. Hansotia MF, Malik G, Memon S. Snakebite. In: Ilyas M, Khan IA, Malik G, Mubasher M, Thaver I, Inam SNB, et al, editors. Community medicine. 5th ed. Karachi: Time Traders, 2000: 1011-29.
2. Hung DZ, Wu ML, Deng JF, Lin-Shiau SY. Russell's viper snakebite in Taiwan: differences from other Asian countries. Toxicon 2002; 40: 1291-8.
3. Jintakoon P, Pochanugool C, Limthongkul S. Venomous snakes in Thailand. In: Gopalakrish-

nakone P, Chuu LM, editors. Snakes of medical importance. Singapore: Venom and Toxin Research Group, National University of Singapore, 1990: 517-64.
4. Kularatne SA. 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: 855-62.
5. Warrel DA. Injuries, envenoming, poisoning and allergic reactions caused by animals. In: Ledingham JGG, Warrel DA, editors. Concise Oxford textbook of medicine. New York: Oxford University Press, 2000: 1889-901.
6. Nayak KC, Jain AK, Sharda DP, Mishra SN. Profile of cardiac complications of snake bite. Indian Heart J 1990; 42: 185-8.
7. Hafeez S, Majeed I. Cardiac arrhythmia as a presentation of snakebite. J Coll Physicians Surg Pak 2004; 14: 48-9.

ภาวะหัวใจเต้นผิดจังหวะอย่างรุนแรงจากงูแมวเซากัด: รายงานผู้ป่วย

ยศพล เตียวจิตต์เจริญ, สุภาพร ภู่พิทยา

งูกัดยังคงเป็นปัญหาสำคัญในแถบชนบทของทวีปเอเชีย พิษของงูแมวเซาที่ทราบกันทั่วไปคือภาวะเลือดออกง่ายและพิษต่อไต นอกจากภาวะทั้งสองแล้ว พิษต่อหัวใจพบได้น้อยมากในผู้ป่วยที่ถูกงูแมวเซากัด ผู้เขียนได้รายงานผู้ป่วยชายถูกงูแมวเซากัดและเกิดหัวใจหยุดเต้นจากภาวะหัวใจเต้นผิดจังหวะอย่างรุนแรง ผู้ป่วยได้รับการรักษาอย่างเร่งด่วนโดยการช็อกหัวใจและให้ยา Amiodarone หลังจากนั้น Antivenom สำหรับงูแมวเซาถูกให้เพื่อแก้ไขภาวะเลือดออกง่าย