An Unusual Case of Acute Asthma after Snake Bite



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ABSTRACT

BACKGROUND

Although the cytolytic, neurotoxic and haemolytic actions of snake venoms are well known, the ability of snake venom to induce asthma (as a distinct entity from just difficulty in breathing) is not previously reported in the literature.

METHODS

The case records of the patient in the index case and a review of existing literature using online search via google, medline and pubmed with the following key words Snake bite; envenomation and acute asthma were utilized.

RESULT

We report the case of a 42 year Nigerian fisherman, not a previously known asthmatic, who was bitten by a snake and developed acute asthma in 30 minutes.

CONCLUSION

Acute Asthma after snake bite in the absence of signs of envenomation is rare. A high index of suspicion is needed in identifying this probable condition in patients exposed to snake bite. There is need for further investigation into the pathogenesis for this presentation.

Keywords: Snake bite; Envenomation; Acute Asthma.

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INTRODUCTION

Snake bite is common occurrence in Nigeria especially in the rural areas [1], [2]. Many species of snakes abound in Nigeria just like in many other parts of the world [2], especially in tropical and subtropical areas where snake bites are a serious problem.

It has been estimated that five million snakebite cases occur every year, resulting in about 100,000 deaths annually worldwide [3]. The recent categorization of snake envenoming as a neglected tropical disease (NTD) is an important advance that hopefully will result in the wider recognition and allocation of resources to this condition. Particularly since death from snake envenoming is preventable and snake antivenom is a very effective treatment when correctly administered [4].

The venomons snakes in Africa are known to belong to four main families the colubridae, elapidae, viparidae and hydrophidae, but in Nigeria, the most common poisonous snakes are the elapidae and viperidae [5].

Snake venoms are used primarily for attack and contain components designed to immobilize prey and facilitate their digestion.

Over 95% of the dry weight of most venom is polypeptides which include enzymes, toxins, and small peptides, each class being capable of modulating the physiological response of envenomed animals. More than 20 enzymes have been detected in snake venom and 12 are found in majority of venoms [6]. Hyaluronidase is present in all snake venoms facilitating the distribution of other venom components throughout the tissue of the prey [6].

Elapids account for the vast majority of deaths worldwide, particularly as a result of toxins that act at the neuromuscular junction. Systematic manifestations occur after about 30 minutes to 1 hour and include ptosis, external ophthalmoplegia, dysphagia, salivation followed by general paresis and respiratory failure. These presynaptically acting toxins exhibit phospholipase A_2 activity [7].

Cardiotoxins which cause augmentation of myocardial contractions at low concentration have been identified from cobra venoms [8]. Crotamine rattle snake venoms have a specific and unique effect on the sodium channel of excitable membrane [8]. Phospholipase A_2 neurotoxins (e.g. beta bungarotoxin) have been shown to block potassium channels while a component from the venom of the rattle snake (crotalusatrox) affects calcium channels [9].

Haemorrhagic symptoms are a frequent accompaniment of bites by vipers and of some venomous colubrids [10]. Venom procoagulants activate prothrombin, and factor V and X. Some venom components have direct thrombin-like effect. Thrombocytopenia may occur and platelet function may be affected. Spontaneous systematic bleeding is caused by haemorrhagins which damage the vascular endothelium. Massive intravascular haemolysis leading to renal failure has also been reported [11].

The review of the literature on the clinical presentation and mechanism of envenomation with snake bite indicates a rarity of anaphylaxis features and even more the features of acute asthma attack. It is on this background that this case report is presented to draw attention to this rare manifestation after snake bite.

CASE REPORT

M.A is a 42year old male Nigerian fisherman, previously fit with no past medical history, who presented at the Accident and Emergency Department of our hospital with

breathlessness, wheezing, diaphoresis, agitation and inability to complete sentences 30minutes after he was bitten by a snake close to the bank of the river he had done fishing. The bite was on the left ankle. Apart from use of tourniquet below the knee no other form of treatment was given.

His temperature was 37.4°C, the pulse rate was 118 per minute and his respiratory rate was 28 breaths per minute. The blood pressure was 140/80mm/Hg. Examination of the site of bite showed two fang marks with blood on the skin. The site of bite showed significant local swelling. He was alert with normal mental status. The rest of the central nervous system was normal. Apart from tachycardia, the cardiovascular system was normal. The breath sounds were decreased bilaterally with widespread wheezes. Examination of the abdomen was negative. His peak expiratory flow rate (PEFR) using a Wright peak flow meter gave his best reading as 130L/min which was less than 40% of predicted value [12]. A chest radiograph ruled out infiltrates, atelectasis and pneumothorax. He was hypoxic and hypocapnoeic on room air at Pa02 12Kpa (90mmHg) and PaC02 45Kpa (34mmHg) respectively. His PCV was 38% and white cell count was 4.3 x 10⁹/L, erythrocyte sedimentation rate was 48mm/hr. his serum sodium was 141mmol/L, potassium 3.6mmol/L, chloride 111mmol/L, urea 3.5mmol/L, creatinine 94µmol/L. His partial thromboplastin time was 42.4 seconds and prothrombin time was 12.3 seconds all within normal limits. The ECG showed sinus tachycardia with no ventricular strain nor right axis deviation. A diagnosis of Systemic inflammatory response syndrome (SIRS) complicating snake bite was made.

In view of the severe bronchoconstriction, salbultamol was given by oxygen-driven nebulization with aminophylline in normal saline infusion as it was confirmed he was not previously on any methylxanthine. Intravenous hydrocortisone was also given. He continued to deteriorate with the development of a 'silent chest'. He had killed

the snake and came with it to the emergency room but it was difficult for us to identify the specie of snake. (see figure 1).

With the marked local swelling and pain it was clear there had been envenomation. Tetanus toxoid was given since there was no evidence of coagulopathy. Polyvalent antivenom (5 vials of 10mls) was given in 1 litre of normal saline infusion over 2hours. About an hour later he was observed to be less breathless but still wheezing with peak expiratory flow rate best of three readings of 198L/min. This was still less than 40% of predicted value. His SaO₂ had increased from 82% to 88%. Four hours later the antivenom was repeated after which he started to make significant recovery. By the third day of admission the best of three measurement of his PEFR was 520L/min. He was discharged on the fifth day.

DISCUSSION

This case was found worth reporting as to the best of our knowledge, no such report is in the literature. The closest to this report is that by Prescott and Potter who reported bronchial asthma in snake handlers from aerosolized snake venom as against snake envenomation [13].

Our patient did not show any signs of coagulopathy nor platelet dysfunction. Neither did he exhibit any signs to suggest neurotoxicity. He, however, had very significant local swelling with acute bronchoconstriction. Even though he did well and was discharged over nine months ago, this patient in subsequent follow-up clinic visits, has continued to have recurrent episodes of wheezing, breathlessness and chest tightness. We are at a loss as to which of the venom's peptide which usually include enzymes, toxins and small peptides could be responsible for this presentation. The identification of such substance, however, awaits further study.

Figure1: Unidentified snake specie.



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