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CASE REPORTS

STICK OUT YOUR TONGUE: LINGUAL MYOKYMIA A SIMPLE NEUROTOXIN TEST AS A DIAGNOSTIC MARKER AFTER SNAKE BITE IN A CHILD

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ABSTRACT

Crotalidae, commonly known as pit vipers are venomous snakes that include copper heads, water moccasins, and rattlesnakes which inhabit most of North America. Crotalidae possess hollow anterior fangs that deliver venom into subcutaneous tissue, though not all bites result in envenomation.[1] Whether venomous or dry, bites can lead to inflammation, infection, and anxiety.[4] Systemic symptoms, which indicate that venom was delivered, include nausea, vomiting, hypotension, coagulopathy, and neurotoxicity.[4] We present a case of lingual myokymia following a rattlesnake bite in a 9-year-old male, highlighting a distinctive method for rapidly identifying systemic neurotoxic effects post-envenomation. Upon arrival at the pediatric emergency department, he presented with local swelling and four puncture wounds at his left medial malleolus identified as the bite site. Despite unremarkable lab findings, lingual myokymia was noted on physical exam. The patient required two rounds of antivenom with a second dose administered after progressive symptomatology. In addition to examination of the bite site, a simple oral exam which includes an assessment of tongue movement, can help differentiate between venomous bites and dry bites, providing critical information for clinical decision-making in both urban and wilderness contexts. This easily teachable method can empower layperson and clinicians alike to make informed evacuation and clinical decisions based on early identification of systemic neurotoxic effects.

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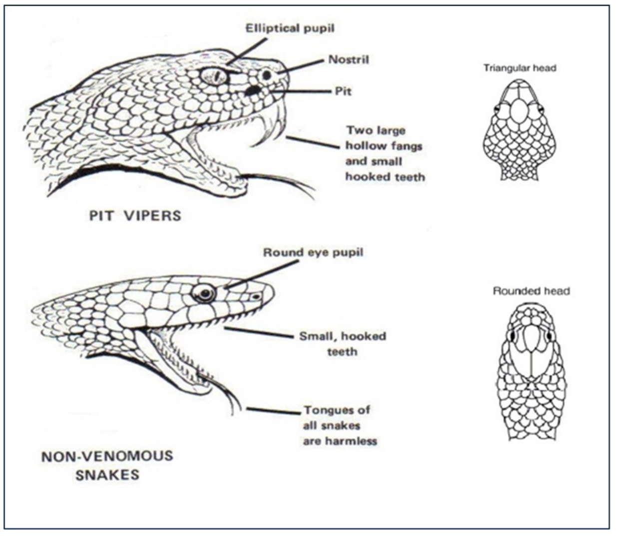
Introduction

Most snakebites in the United States are from pit vipers[1] where 7,000 people receive treatment annually for pit viper envenomation.[2] Pit vipers, or Crotalidae, include copperheads, water moccasins, and rattlesnakes, species that are widespread across North America. Since venom production is metabolically taxing, a proportion of snakebites are dry, where no venom is injected.[3] One study conducted in Central California demonstrated that only about 10% of Crotalidae bites presenting for care were dry bites and did not require anti-venom.[4] Most incidents occurred in the spring and summer, peaking in June.[4]

Crotalidae venom contains a potent mixture of proteolytic enzymes, hemotoxins, and neurotoxins (including phospholipase A2 toxins) that can result in local tissue destruction, systemic coagulopathies, and neurotoxic effects.[1] Children are at high risk of snakebites, often being bitten while playing outside. Due to their overall lower body weight and immature immune systems, envenomation in children tends to be more severe.[3]

The severity of envenomation can be classified as dry, minimal, moderate, or severe. Dry bites involve fang marks with no systemic or local effects, while minimal envenomation results in localized pain and swelling. Moderate cases exhibit progressive swelling and non-life-threatening systemic effects, whereas severe envenomation may cause angioedema, thrombocytopenia, and shock.[5] Antivenom therapy is typically recommended for moderate to severe

Figure 1: Pit Viper Anatomy vs Non-Venomous Snake venomous snakes have larger and curled fangs for injecting venom into subcutaneous tissue[14].



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cases. Unnecessary administration of antivenom can lead to adverse reactions due to the introduction of heterologous immunoglobulins.[3]

Laboratory evaluation should begin with a comprehensive initial workup, including a complete blood count, prothrombin time, partial thromboplastin time, fibrinogen levels, creatine kinase, basic electrolyte panel, and urinalysis to monitor for the potential development of proteinuria or hematuria. Neurological status should be assessed regularly to monitor for signs of neurotoxicity. In all Crotalidae species, antivenom therapy remains the cornerstone of treatment as it effectively neutralizes venom components.[6] Additionally, the bite site should be examined frequently for the progression of edema, blister formation, and signs of compartment syndrome.[7]

Myokymia, a rippling muscle movement from motor neuron bursts, differs from fasculations, which activate individual motor units.[8] Myokymia from snakebites is typically localized near the bite site or facial muscles. We describe a rare case of lingual myokymia following a rattlesnake bite in a child.

Case Report

A previously healthy, fully vaccinated, 9-year-old male with no significant past medical history presented to pediatric emergency department (PED) after sustaining a rattlesnake bite inferior to the left medial malleolus. He was transported to the PED by emergency medical services (EMS), remaining stable throughout transport. Upon arrival, physical examination revealed localized swelling, and four-puncture wounds at the bite site, consistent with a multi-fang strike. The patient reported tingling in his distal extremities, accompanied by a "bitter" taste in his mouth. EMS had outlined the initial area of swelling (Figure 2).

Figure 2: Bite site, left medial malleolus, line drawn by EMS

On initial exam the patient was alert, oriented, with no signs of altered mental status. Retropharyngeal exam to evaluate for airway edema revealed involuntary lingual myokymia. Pulses in the left lower extremity remained palpable. A perimeter was drawn around the swelling at the bite site continuously every 20-30 minutes. He had full range of motion of his left ankle with strength 5/5 in plantar flexion and extension. While he did report a tingling sensation in both his upper and lower extremities, light touch and pinprick sensation remained intact throughout his extremities. Localized edema around the left ankle demonstrating gradual progression over the course of the PED stay.

Initial laboratory workup was unremarkable for significant electrolyte derangement, thrombocytopenia, or coagulopathic abnormalities. The patient's creatine kinase was elevated at 865 U/L (reference range 39-293 U/L), without evidence of renal impairment. An electrocardiogram (ECG) demonstrated normal sinus rhythm with no arrhythmias and normal intervals. Snakebites can cause atypical ECG changes, including T-wave abnormalities, ST elevation, and atrial fibrillation.[9] The snakebite occurred at approximately 15:30, with the first dose of antivenom (10 vials of ANAVIP) administered at 17:05. The patient was subsequently admitted to the pediatric intensive care unit with serial assessments and laboratory tests every six hours. His treatment plan included pain management with acetaminophen, limb elevation, continued monitoring for signs of systemic toxicity, and intravenous fluid administration.

During his stay, the patient experienced an elevation in prothrombin time, from 15.8 to 16.2 seconds (reference range: 12.7–15.8), with a corresponding increase in INR to 1.17 (reference range: 0.86–1.14). Despite initial treatment, his symptoms progressed, with increasing facial paresthesia, worsening myokymia, and edema progression. A second dose of antivenom was administered approximately eight hours after the initial dose.

Following the second dose, the patient's symptoms improved. The patient was discharged with instructions for follow-up with his pediatrician for repeat laboratory assessments, including a complete blood count, basic metabolic panel, and fibrinogen levels. To this date we are not aware of further complications.

Discussion

Oral examination is essential in snakebite assessment to check airway patency and neurotoxic signs like lingual myokymia. Asking the patient to protrude their tongue is a simple yet effective way to detect systemic neurotoxic manifestations.

An illustrative case includes a 14-year-old boy who exhibited myokymia of the deltoid, lower face, and calf muscles following a rattlesnake bite.[8] Neurotoxic snakebite envenomation may begin with ophthalmoplegia and ptosis and can progress to facial and vocal cord paralysis.[10] Respiratory failure from diaphragmatic paralysis requires urgent intervention.[10] While involuntary movements, such as lingual myokymia, are uncommon, they may represent the interaction of venom within neuronal axons.[11]

Opioid analgesics should be avoided as to not mask the neurotoxic effects of the venom.[12] Pain control was achieved in this patient by use of acetaminophen. Wound infections following pit viper bites are rare, and therefore, it is not recommended to use antibiotics prophylactically.[12] Tetanus status however should be inquired and administered if not up to date.

Conclusions

A thorough physical examination searching for muscle tremors, including inspection of the oral cavity and specific assessment for lingual myokymia through tongue protrusion, should be incorporated as a part of the standard approach following snakebites. Absence of lingual myokymia does not exclude envenomation, however its detection enables early recognition and initiation of treatment.

Compliance with Ethical Standards

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