Neurotoxic Snakebite Presenting with Abdominal Pain: A Case Report

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ABSTRACT

An early sign of neurotoxic snakebite envenomation is abdominal pain. We present a 14-year boy from a rural area who was admitted with acute abdominal pain, occurring from midnight to early morning, followed by neuroparalytic symptoms. After ruling out other causes, the boy was diagnosed with occult snakebite envenomation, likely from a krait, as it is typically painless, lacks fang marks, and often occurs at night. Similar cases have been reported, where suspected occult snakebites responded to anti-snake venom (ASV) treatment. Clinicians should consider snakebite envenomation, particularly in patients from rural areas presenting with acute abdominal pain during the rainy season, and monitor for neurotoxicity, as timely ASV administration leads to faster recovery.

Key Words: *Anti-smoke venom, Snakebite, Abdominal pain, Krait*.

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INTRODUCTION

Though a medical emergency, snake envenomation is still considered an overlooked tropical disease by the World Health Organisation (WHO). South Asia accounts for about 70% of all snakebite deaths worldwide.[1,2] Over 40,000 cases and 8,200 fatalities are reported in Pakistan each year. Pakistan is home to 72 distinct species of snakes, 40 of which are poisonous. The "Big Four" snakes in the region—the common krait, Russell's Viper, saw-scaled viper, and Indian cobra—are the main cause of snakebite mortality. Unfortunately, there is only one polyvalent anti-snake venom (ASV) available.[2]

Snakebites commonly occur during the monsoon months. The actual number of snakebite deaths is unclear because they are frequently under-reported in rural regions.[2,3] People from agricultural families, those residing in clay huts, where they often sleep on the ground, and children are more likely to be bitten by snakes.[3,4]

Neurotoxic snakebites are often missed or diagnosed late due to a lack of history of snakebites and absent fang puncture marks.[1] The presenting complaint may be abdominal pain as early as 30 minutes after the bite. This is followed several hours later (8 to 19 hours) by the onset of neurological symptoms including ptosis, foamy saliva, slurred speech, respiratory failure, and descending paralysis.[1,5]

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We report a case of an occult snakebite presenting with abdominal pain followed by paralysis and respiratory distress.

CASE REPORT

A 14-year boy from Talagang developed crampy abdominal pain one day back on waking up in the morning. A few hours later, he felt weakness in his lower limbs followed by weakness in his upper limbs, and difficulty opening his eyes. He had a history of sleeping on a mattress on the floor. He was taken to THQ Hospital, Talagang where he was drowsy and in respiratory distress, so he was moved to the tertiary care hospital with oxygen support.

On presentation, his initial Glasgow Coma Scale score was 8/15, blood pressure was 100/70 mmHg, pulse was 140/minute, random blood sugar was 112 mg/dl, and oxygen saturation was 60% at room air.

Physical examination revealed obvious respiratory distress, bilateral ptosis, positive left Babinski sign, sluggishly reactive pupils, excessive mouth frothing, and urinary incontinence. Due to ambiguous clinical presentation, there was a suspicion of snakebite with respiratory failure. He was intubated, given ASV, pyridostigmine, and atropine and was referred to our hospital due to the non-availability of a ventilator.

At our hospital, he was admitted to the Intensive Care Unit (ICU) and put on a ventilator on synchronised intermittent mandatory ventilation (volume control) with pressure support mode. He was sedated and paralysed. There was a healing laceration below his right little toe. No fang-puncture marks were found. Bowel sounds were sluggish and there was bilaterally decreased lung air entry on chest auscultation.

Initial investigations are shown in Table I. Arterial blood gases (ABGs) at presentation showed type 2 respiratory failure. Cere-

brospinal fluid studies including routine examination, culture, and Herpes Simplex Virus DNA PCR were unremarkable. Bedside electroencephalogram showed paroxysmal discharge of slow waves (delta and theta waves). CT scan brain was normal. Further investigations included nerve conduction studies (NCS) and electromyography. Repetitive nerve stimulation (RNS) showed a decremental response of more than 10%. Acetylcholine receptor antibodies and anti-muscle-specific kinase antibodies were negative.

He was weaned off mechanical ventilation and extubated the next day. His conscious level had improved. However, he remained tachycardic and tachypnoeic and required non-invasive ventilation *via* bilevel positive airway pressure (BiPAP). He was given an empirical trial of pyridostigmine after which all weakness reversed within three days.

The initial differential diagnoses included Guillain-Barre syndrome (GBS), botulism, myasthenia gravis (MG), and snakebite. GBS and botulism were ruled out on NCS and electromyography. Although the RNS was suggestive of MG, the acetylcholine receptor antibodies and anti-muscle-specific kinase antibodies were negative, which along with the unusual age group of the patient, negated the diagnosis of MG.

Despite absent fang marks and a witnessed event, a neurotoxic snakebite was suspected. There was a favourable history of sleeping on the floor, presentation during monsoon season, and sudden onset of neurological symptoms after abdominal pain. The timely administration of ASV slowed down the progression of neurological symptoms. At the time of discharge, he was vitally stable with no neurological deficit. Furthermore, once pyridostigmine was discontinued, his RNS test was repeated, which was normal.

Table I: Initial laboratory investigations.

Investigations	Patient's results	Reference ranges
Total leucocyte count	9.1 × 10^3/ ul	4-11 × 10^3/ul
Haemoglobin	13.5 g/dl	13-17 g/dl
Platelet count	185 × 10^3/ul	150-400 × 10^3/ul
Sodium	133 mmol/ L	135-145 mmol/L
Potassium	4.3 mmol/L	3.5-5.5 mmol/L
C-reactive protein	55.6 mg/L	0-7.5 mg/L
PT/APTT/INR	15 s/ 67 s /1.4	10-12 s/22-32 s/1.1
Serum lipase	20.1 U/L	10-60 U/L
Serum urea	2.8 mmol/L	1.7-8.3 mmol/L
Serum creatinine	42 umol/L	53-105 umol/L

DISCUSSION

A case series reported by Deopa *et al*. from India described neuroparalytic snakebites that presented as severe gastrointestinal pain in young patients. During the rainy season, four children from a rural background were admitted one after the other over the course of a month. Their initial complaint was severe abdominal pain, followed hours later by bulbar symptoms, breathing problems, and limb weakness. Three of them required ventilatory support. Out of the patients who received ASV, the patients who got it sooner recovered earlier.[1]

Mehta *et al*. reported three cases where young males had severe paralysis as a result of occult elapid snakebites.[6] An intriguing observation in all three cases, which is similar to the present case, was the fact that the first complaint described by each patient was abdominal pain followed by neuromuscular paralysis. None of them had any obvious history of being bitten by a snake or visible fang marks. Their symptoms began during night time. All three patients slept on the floor. As each patient was a diagnostic challenge, extensive diagnostic tests were performed which remained inconclusive. They were presumed to be occult snakebites as a diagnosis of exclusion and all of them showed dramatic clinical improvement after receiving ASV, neostigmine, and supportive treatment.[6]

Neuroparalysis can result from elapids (krait or cobra bites).[1] Cobra bites are identified sooner because they typically occur outdoors, are accompanied by fang marks, and cause notable local inflammation at the bite site. Kraits, on the other hand, are nocturnal species with a propensity to attack people while they are asleep at night.[3] Early morning neuroparalytic syndrome may ensue from this condition. In children who were healthy the previous evening, this condition describes painless bites that happen indoors during the rainy season without any accompanying signs of inflammation. They have vague "gastrointestinal" symptoms around midnight, such as nausea, vomiting, and abdominal discomfort, which are followed by paralysis.[5,7]

It is imperative to maintain a high degree of suspicion of snakebite when patients present with abdominal pain followed by neuromuscular weakness, regardless of the absence of a history of snakebite and fang marks on the body. A collateral history of sleeping on low-lying mattresses, rural background, and presenting during monsoon season may aid in diagnosis. An early initiation of therapy with ASV will enhance recovery in such cases.

PATIENT'S CONSENT:

Informed consent was obtained from the patient to publish the data concerning this case.

COMPETING INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

AM, AS, SA: Conception, drafting, designing, acquisition, analysis, and interpretation of data for important intellectual content.

All authors approved the final version of the manuscript to be published.

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