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# A rare case of strychnine poisoning by consumption of *Strychnos nuxvomica* leaves

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#### ARTICLE INFO

#### ABSTRACT

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### **1. Introduction**

Strychnine is one of the oldest poisons known to man. The source of this potent toxin is the *Strychnos nux–vomica* plant, which actually contains two distinct alkaloids– strychnine and brucine. Although principally found in the seeds, these alkaloids can be isolated in varying concentrations from virtually all parts of the plant including the bark, leaves and roots<sup>[1]</sup>. Once consumed, strychnine produces powerful and uncontrollable muscle contractions that can eventually result in death.

## 2. Case report

A 22-year-old male with no premorbidities, presented with severe myalgias and backache of acute onset. He also complained of one episode of backward arching of the entire body, consistent with opisthotonus. On repeated questioning, he admitted to having consumed approximately 25 leaves of kasarka (the local name for *Strychnos nuxvomica*) three hours prior to presentation with suicidal

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intent. Unconsumed leaves from the same plant were later produced for identification and confirmed to be those of the *Strychnos nux–vomica* plant.

A 22-year-old male presented with severe myalgias and backache of acute onset, as well as

one episode of opisthotonus. On repeated questioning, he admitted to having consumed leaves

of Strychnos nux-vomica with suicidal intent. He was treated conservatively with intravenous

diazepam and analgesia and made a complete recovery. Strychnine poisoning is rarely

encountered and poisoning by consumption of leaves is even rarer. This case demonstrates the

potent toxicity of strychnine as well as the effectiveness of therapy when initiated early.

General physical and systemic examinations were unremarkable except for severe thigh and calf muscle tenderness. Routine laboratory parameters showed elevated serum creatine kinase levels (2 189 U/L), with normal renal function tests. He was treated conservatively with intravenous diazepam and analgesics. Except for one episode of transient opisthotonus immediately after presentation, he made a rapid recovery and was discharged.

## 3. Discussion

While accidental poisoning by exposure to rodenticide containing strychnine is only occasionally reported<sup>[2]</sup> in Western medical literature, such cases are frequently encountered in the Asian populations<sup>[3]</sup> where strychnine derivatives are still used in various forms as a part of traditional medicine<sup>[4–6]</sup>. In India, the majority of cases are related to intentional self–harm. Strychnine is rapidly absorbed from all routes<sup>[7]</sup>. Clinical features of toxicity may develop as early as five minutes after inhalation, 30 minutes after ingestion<sup>[8]</sup> and upto 24 hours after transdermal exposure<sup>[9]</sup>. Ingestion of less than 10 mg in a child<sup>[10]</sup> and 16 mg in an adult have been reported to be fatal. Once



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absorbed, strychnine antagonizes glycine<sup>[11]</sup>, an important inhibitory neurotransmitter in the spinal cord, brainstem and higher centers. This directly acts at the post-synaptic receptors of the spinal motor neuron<sup>[12]</sup> resulting in loss of inhibitory tone and producing characteristic muscle spasms, known as spinal seizures as the patient remains fully alert<sup>[2]</sup> despite generalized convulsive movements and can be provoked by even minimal stimulation<sup>[13]</sup>. Other proposed mechanisms of toxicity include agonism of excitatory NMDA receptors<sup>[14]</sup> and antagonism of GABA<sup>[15]</sup>, another inhibitory neurotransmitter in the central nervous system. Involvement of thoracic and abdominal musculature resulting in respiratory paralysis is the commonest cause of death, and few patients survive more than five convulsive episodes without treatment<sup>[16]</sup>. Notable complications of strychnine poisoning with uncontrolled muscle spasms include hyperthermia<sup>[12]</sup>, severe lactic acidosis and rhabdomyolysis. Important differential diagnosis include tetanus, epilepsy, dystonic drug reactions, infections of the neck, hypocalcemia, picrotoxin exposure and psychogenic disorders. Upto 80 percent of ingested strychnine is eliminated through hepatic metabolism<sup>[17]</sup> whilst the remaining 20 percent is excreted in the urine<sup>[7,18]</sup>. This rapid elimination means that the prognosis is generally favourable in patients surviving beyond five hours from the onset of symptoms<sup>[18]</sup>. Once diagnosed, treatment modalities include initial stabilization with oxygen, avoidance of all extraneous stimuli, control of muscle spasm with benzodiazepines and barbiturates, and airway management including endotracheal intubation. Haemodialysis has not proven effective in enhancing the elimination of strychnine<sup>[19]</sup>. Intravenous fluids should be administered to maintain a brisk urine output (>1 mL/kg/h) as rhabdomyolysis[20,21], metabolic acidosis and acute renal failure<sup>[22,23]</sup> are wellknown complications. Fortunately in our case, such complications were not seen and our patient made a full and uneventful recovery.

As our case demonstrated, acute and severe strychnine poisoning can occur from consumption of any part of the plant, including the leaves. To the best of the authors' knowledge, this is the first case of strychnine poisoning reported in medical literature following the consumption of leaves of the *Strychnos nux–vomica* plant.

#### **Conflict of interest statement**

We declare that we have no conflict of interest.

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