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REVIEW

Strychnine poisoning in children

Intoxicación por estricnina en edad pediátrica

Ingrid Narel Davalos Zuleta[1]
Alexandra Andreé Quiroga Gamarra[2]
Antonio Viruez Soto[3]
Freddy Ednildon Bautista-Vanegas[4]
Sara Milca Robles-Nina[5]
Jhossmar Cristians Auza-Santivañez[6]

[1] Hospital del Norte El Alto. Departamento de Terapia Intensiva Pediátrica. La Paz, Bolivia.
[2] Hospital del Norte El Alto. Departamento de Pediatría. La Paz, Bolivia.
[3] Hospital del Norte El Alto. Departamento de Apoyo Crítico. La Paz, Bolivia.
[4] Kliniken Beelitz GmbH Neurologische Rehabilitationsklinik: Beelitz Heilstätten, Brandenburg, DE. Germany.
[5] Hospital de Segundo Nivel Walter Khon. Oruro, Bolivia.
[6] Hospital del Gran Chaco Fray Quebracho. Instituto Académico Científico Quispe-Cornejo. La Paz, Bolivia.

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Corresponding Author: Jhossmar Cristians Auza-Santivañez

ABSTRACT

Strychnine is a highly toxic alkaloid obtained from Strychnos nux-vomica that acts as a competitive antagonist of glycine receptors in the central nervous system, producing severe muscle spasms, generalized seizures, and respiratory compromise. Although cases of strychnine poisoning are currently uncommon, they remain a medical emergency because of their rapid progression and high mortality if not recognized and treated promptly. This article reviews the main toxicological characteristics of strychnine, including its mechanism of action, clinical manifestations, diagnosis, differential diagnosis, and current therapeutic management, emphasizing supportive treatment and early intensive care. The available evidence highlights the importance of rapid diagnosis, prompt airway protection, seizure control, and multidisciplinary management to improve clinical outcomes. Preventive strategies aimed at reducing accidental and intentional exposure to this toxic agent are also discussed.

Keywords: Strychnine; acute poisoning; neurotoxicity; seizures; emergency medicine; intensive care.

RESUMEN

La estricnina es un alcaloide altamente tóxico obtenido de Strychnos nux-vomica, cuyo mecanismo de acción consiste en el antagonismo competitivo de los receptores de glicina en el sistema nervioso central, lo que ocasiona espasmos musculares intensos, convulsiones y compromiso respiratorio. Aunque las intoxicaciones por estricnina son poco frecuentes en la actualidad, continúan representando una emergencia médica debido a su rápida evolución y elevada mortalidad en ausencia de un tratamiento oportuno. El presente trabajo revisa las principales características toxicológicas de la estricnina, incluyendo su mecanismo de acción, manifestaciones clínicas, diagnóstico, diagnóstico diferencial y manejo terapéutico, con énfasis en el tratamiento de soporte y los cuidados intensivos. La evidencia disponible destaca la importancia del reconocimiento precoz, el control inmediato de la vía aérea y de las convulsiones, así como el abordaje multidisciplinario para mejorar el pronóstico. Asimismo, se resalta la necesidad de fortalecer las estrategias de prevención para disminuir la exposición accidental e intencional a este agente tóxico.

Palabras clave: Estricnina; intoxicación aguda; neurotoxicidad; convulsiones; medicina de urgencias; cuidados intensivos.

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INTRODUCTION

Strychnine is an alkaloid extracted from the tree Strychnos nux vomica, also known as Saint Ignatius bean, a plant native to the tropical forests of Southeast Asia, especially India. (1,2)

It appears as a white crystalline powder with an extremely bitter taste (3). Its main use since the XVI century has been as a rodenticide. (1) It has also been used as a cathartic in herbal medicine and as a respiratory, circulatory, and digestive stimulant. (1,4)

It is frequently used to adulterate illicit drugs such as cocaine and heroin; additionally, it is involved in cases of poisoning, both voluntary and accidental. Recently, the antitumor effect of this alkaloid in human hepatocellular carcinoma (HCC) has been studied. (1)

The potent toxicity of strychnine is due to its action as a competitive antagonist of the inhibitory neurotransmitter glycine, mainly in the spinal cord and brainstem. This causes uncontrolled stimulation of postsynaptic neurons and diffuse involuntary muscle contractions. Clinically, this manifests with severe muscle spasms (including opisthotonos, risus sardonicus, and trismus), while the patient remains conscious and oriented. (5)

Symptoms usually appear 15 to 30 minutes after oral ingestion, and more rapidly when exposure is via intranasal or intravenous routes. (4) Strychnine exhibits accelerated systemic distribution, is metabolized in the liver, and has an elimination half-life of approximately 10 to 16 hours. (4)

The estimated lethal dose in adults ranges from 5 to 30 mg, and from 5 to 10 mg in children. Death commonly occurs due to respiratory paralysis secondary to intense spasms of the respiratory muscles. Although its toxicity is high, cases of strychnine poisoning are uncommon globally (3); for example, in 2015, only 72 exposures and one death were recorded in the United States (5).

Its use is generally restricted in many countries for specific purposes such as the elimination of stray animals by authorized personnel. (5)

Treatment is primarily supportive, with benzodiazepines for the control of muscle spasms and management of complications such as rhabdomyolysis, hyperthermia, and acidosis. (6)

In refractory cases, anesthetics or neuromuscular blockers can be used. (6) Currently, new therapeutic approaches, such as anti-strychnine immunotherapy, are being investigated to reduce or prevent its toxic effects. (7)

METHODS

A narrative review of the scientific literature on strychnine poisoning was conducted, with emphasis on the pediatric population, pathophysiological mechanisms, clinical manifestations, diagnosis, differential diagnosis, treatment, and prevention.

The bibliographic search was conducted between May and July 2025 in the PubMed/MEDLINE, Scopus, EMBASE, and Google Scholar databases, complemented by consulting documents available in SciELO and the StatPearls knowledge base, due to their relevance to clinical toxicology.

The following search terms in English and Spanish were used, individually and combined using Boolean operators:

("strychnine" OR "strychnine poisoning" OR "strychnine toxicity") AND ("children" OR "pediatric" OR "paediatric" OR "acute poisoning" OR "neurotoxicity" OR "seizures")

and

("strychnine" OR "strychnine poisoning") AND ("children" OR "pediatrics" OR "acute poisoning" OR "seizures" OR "neurotoxicity")

Original articles, narrative reviews, case reports and series, clinical guidelines, and reference resources published in English or Spanish between 1995 and 2025 were included, addressing epidemiological, pathophysiological, clinical, diagnostic, or therapeutic aspects related to strychnine poisoning. Highly relevant classic publications were also included when they provided fundamental information on the compound's toxicity.

Congress abstracts without full text, duplicate publications, articles not related to human strychnine poisoning, and documents with insufficient information for the objectives of the review were excluded.

The selected information was analyzed through narrative synthesis and organized into the following thematic axes: pathophysiology, epidemiology, clinical manifestations, diagnosis, differential diagnosis, treatment, and prevention, with the aim of integrating the available evidence and providing an updated overview of the clinical management of this intoxication.

RESULTS AND DISCUSSION

Strychnine is an alkaloid extracted from the tree Strychnos nux vomica, also known as Saint Ignatius's bean, a plant native to the tropical forests of Southeast Asia, especially India. (1,2)

It appears as a white crystalline powder with an extremely bitter taste (3). Its main use since the XVI century has been as a rodenticide. (1) It has also been used as a cathartic in herbal medicine and as a respiratory, circulatory, and digestive stimulant. (1,4)

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The potent toxicity of strychnine is due to its action as a competitive antagonist of the inhibitory neurotransmitter glycine, mainly in the spinal cord and brainstem. This causes uncontrolled stimulation of postsynaptic neurons and diffuse involuntary muscle contractions. Clinically, this manifests with severe muscle spasms (including opisthotonos, risus sardonicus, and trismus), while the patient remains conscious and oriented. (5)

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Strychnine has an accelerated systemic distribution, is metabolized in the liver, and has an elimination half-life of approximately 10 to 16 hours. (4)

The estimated lethal dose in adults ranges from 5 to 30 mg, and from 5 to 10 mg in children. Death commonly occurs from respiratory paralysis secondary to severe spasms of the respiratory muscles. Although its toxicity is high, cases of strychnine poisoning are uncommon globally (3); for example, in 2015, only 72 exposures and one death were recorded in the United States (5).

Its use is generally restricted in many countries for specific purposes such as the elimination of stray animals by authorized personnel. (5)

Treatment is mainly supportive, with benzodiazepines for control of muscle spasms and management of complications such as rhabdomyolysis, hyperthermia, and acidosis. (6)

In refractory cases, anesthetics or neuromuscular blockers may be used. (6) Currently, new therapeutic approaches, such as anti-strychnine immunotherapy, are being investigated to reduce or prevent its toxic effects. (7)

Strychnine has been used as an analeptic in human and veterinary medicine, in addition to functioning as a bitter agent, cathartic, tonic, and circulatory, respiratory, and digestive stimulant. However, it is considered ineffective and dangerous for these purposes. It has also been employed in the treatment of impotence, sleep apnea, and nonketotic hyperglycinemia, although its efficacy in these conditions has not been established. (4)

Commercial preparations for the public contain between 0.3% and 0.5% strychnine, while those used by licensed exterminators can have concentrations greater than 5%. (4)

The absorption of strychnine is rapid, whether by the nasal, gastrointestinal, or intravenous route, distributing between plasma and erythrocytes, with approximately 50% of the compound diffusing into tissues within the first 5 minutes. (5,8)

The central nervous system does not accumulate to higher concentrations than other tissues. Its metabolism occurs in the liver through microsomal enzymes, predominantly cytochrome P-450 monooxygenase, and the main metabolite is strychnine-N-oxidase. (5) Excretion is primarily renal, with between 10% and 20% of the dose eliminated unchanged in urine during the first 24 hours, with this proportion decreasing as the dose increases. (4)

Strychnine acts as a selective toxic agent on the central nervous system, predominantly exerting its effect on the spinal cord. Its mechanism consists of competitively antagonizing glycine, an inhibitory neurotransmitter, blocking postsynaptic inhibition in motor neurons of the anterior horn of the spinal cord and brainstem. It does not block the depressant effect of GABA, a neurotransmitter of presynaptic inhibitory neurons. (4) By inhibiting glycine, hyperexcitation of muscle groups occurs due to loss of normal inhibition, resulting in excessive activity of motor neurons. (1)

Poisoning with this alkaloid remains uncommon worldwide. For example, in 2015 there were 72 exposures and 1 death in the United States, (5) while in Hong Kong 12 cases were reported between 2005 and 2018 related to "Strychni semen" (maqianzi). (9) In Venezuela, a toxicology service documented approximately 10 events and 1 death in the last 20 years. (1)

The main sources of poisoning include accidental or intentional ingestion of rodenticides, adulteration of illicit drugs such as cocaine, heroin, and amphetamines (often by nasal insufflation or intravenous use), and use in traditional Chinese or Cambodian herbal remedies such as "maqianzi" or "slang nut." Additionally, its use for performance enhancement in sports has been reported, establishing another possible source of poisoning. (5,9)

Determining the exact toxic dose has been complex; however, the estimated minimum toxic dose is 0.2 mg, and toxic blood concentrations range from 0.5 to 90 µg/mL. (3) It is important to note that toxic serum levels as low as 180 ng/mL (equivalent to 0.18 µg/mL) have been recorded, which underscores the variability and complexity of these concentrations. (6)

In humans, the minimum reported lethal dose in children ranges from 5 to 10 mg, and in adults from 30 to 120 mg, with the lowest lethal dose via the intranasal, intravenous, or subcutaneous routes. (3,5) Symptoms of intoxication can appear rapidly, with seizures occurring within 5 minutes after nasal or intravenous exposure, and approximately 15 minutes after oral ingestion. (5,10)

When inhaled, snorted, or injected, symptoms appear rapidly. (5) Prodromal symptoms may or may not be present, and typically last between 30 and 60 minutes; they include restlessness, anxiety, hyperpnea, myalgias, nervousness, tremors, and muscle spasms. (4) The main event, called strychnine tetanus, is characterized by painful and forceful tonic contractions that can affect the trunk and extremities, accompanied by trismus, risus sardonicus, facial congestion, cyanosis, mydriasis, exophthalmos, hypertension, and tachycardia. (4,5)

The patient usually remains conscious and lucid, experiencing “conscious seizures” or episodic muscle contractions without a postictal period. Mental status is preserved until advanced stages, when the sequelae of uncontrolled muscle contractions—such as hyperthermia, acidosis, and other physiological disorders—can alter it.(5,6) Seizures last 1 to 2 minutes, followed by intervals of 5 to 10 minutes. Without treatment, survival after multiple episodes is rare.(5,7)

The diagnosis can be confirmed by detecting the toxic agent in urine, blood, gastric aspirate, and vomitus; in postmortem cases, also in the brain, spinal cord, liver, and kidney.(4)

Clinical monitoring should include parameters such as creatinine, urea nitrogen, electrolytes, calcium, magnesium, sodium, potassium, bicarbonate, phosphorus, creatine kinase, and arterial blood gases.(4)

The differential diagnosis of strychnine poisoning includes conditions such as tetanus, epilepsy, tetany, and overdose with tricyclic antidepressants, theophylline, chlorinated insecticides, amphetamines, or cocaine. Moreover, strychnine poisoning can mimic other poisonings, potentially delaying diagnosis.(1,11)

Treatment is fundamentally symptomatic and supportive, with a primary emphasis on controlling neuromuscular hyperactivity.(5,12)

CONCLUSIONS

Strychnine intoxication continues to be a rare but severe toxicological emergency, characterized by rapid clinical progression and a high risk of life-threatening complications. Early recognition of its clinical manifestations, timely diagnosis, and immediate initiation of supportive measures are the fundamental pillars for improving patient prognosis.

Available evidence indicates that management should focus on controlling neuromuscular hyperactivity, protecting the airway, treating metabolic alterations, and closely monitoring systemic complications, with a multidisciplinary approach when the severity of the condition requires it.

Similarly, it is essential to strengthen prevention strategies through adequate storage, labeling, and control of substances containing strychnine, as well as to promote education for healthcare personnel to facilitate early recognition of this poisoning and optimize clinical decision-making at different levels of care. These measures contribute to reducing associated morbidity and mortality and to improving the quality of care for patients with acute poisoning from this alkaloid.

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CONFLICT OF INTEREST

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AUTHORSHIP CONTRIBUTIONS

Conceptualization: Ingrid Narel Davalos Zuleta, Antonio Viruez Soto.
Data curation: Antonio Viruez Soto.
Formal analysis: Ingrid Narel Davalos Zuleta.
Research: Antonio Viruez Soto, Jhossmar Cristians Auza-Santivañez.
Methods: Antonio Viruez Soto.
Project administration: Alexandra Andreé Quiroga Gamarra.
Resources: Ingrid Narel Davalos Zuleta.
Software: Ingrid Narel Davalos Zuleta.
Supervision: Ingrid Narel Davalos Zuleta.
Validation: Alexandra Andreé Quiroga Gamarra.
Visualization: Alexandra Andreé Quiroga Gamarra.
Writing – original draft: Ingrid Narel Davalos Zuleta, Alexandra Andreé Quiroga Gamarra, Antonio Viruez Soto, Freddy Ednildon Bautista-Vanegas, Sara Milca Robles-Nina, Jhossmar Cristians Auza-Santivañez.
Writing – review and editing: Ingrid Narel Davalos Zuleta, Alexandra Andreé Quiroga Gamarra, Antonio Viruez Soto, Freddy Ednildon Bautista-Vanegas, Sara Milca Robles-Nina, Jhossmar Cristians Auza-Santivañez.