Review Article

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Strychnine Poisoning: Literature Review

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Abstract

Strychnine poisoning is an quite unusual but a serious poisoning in which convulsions are the major threat to life. Convulsions are predominantly noted at the spinal level, and the key to recognizing this poison is observation of convulsive activity in the awake patient without a postictal phase. Successful treatment requires aggressive airway control and treatment of seizures with benzodiazepines or barbiturates. Neuromuscular blockade may be required. Gastrointestinal decontamination is usually indicated in recent acute ingestions but may precipitate convulsions. Recovery from strychnine poisoning is usually complete and rapid if treatment is aggressive.

Keywords: Strychnine; Poisoning; Convulsions.

INTRODUCTION

Strychnine was one of the most famous syntheses in the history by which two chemists won the noble prize (Robinson in 1947 and woodward in 1965) in the field of organic chemistry.[1] It was one, which has turned from medicine to poison through ges. Though it has been discovered in 1818 it has been used to kill dogs, cats and birds in Europe during 1600's.[1] During late 19th and early 20th centuries, it was popularly used as an athletic performance enhancer, recreational stimulant and believed to be cure for alcoholism addiction.[2] It has been familiar with murder mistries[3,4] (Alexander the Great, Jane Stanford cofounder of Stanford university and season 4 Game of thrones king Joffrey) and doping in olympics (1904 and 2016).

Strychnine is extracted from the plant strychnos nux vomica (genus – strychnos, family - loganiaceae) which are found in southern asia (India, Srilanka, East Indies) and Australia. At present, it is being used primarily as pesticide, particularly to kill rats. Devilishly, it was found mixed with street drugs such as LSD, Heroin and Cocaine.

Though strychnine poisonings are rare these days, ingestions (homicidal, suicidal and accidental) still occur in many places. So there is a dire need to know about varied presentations of strychnine toxicity and necessary treatment as this entity doesn't give much time between the consumption and irreversible damage to life. Hereby we would like to present a case series of poisonings and the review of literature regarding the famous rare poisonous substance: The Strychnine (Nux vomica).

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Etiology:

Strychnine poisoning can occur via ingestions, inhalational and intravenous routes. Earlier accidental ingestion were frequently seen as it has been used in many medicines (Used as a respiratory, circulatory and digestive stimulant). By the early 20th centuries, accidental ingestion has reduced a lot. Now a days, strychnine is being used in street drugs (white powder form heroin, cocaine), as pesticide (to kill rats), herbal medicines in some countries like china and combodia.[5,6,7]

MECHANISM OF ACTION

Strychnine acts by competitive inhibition of glycine receptor in the spinal cord. Uncontrollable muscle contractions occurs by negating the inhibitory effects of glycine at the postsynaptic junction predominantly in the spinal cord.[8] Due to these postsynaptic effects, patients will parade ''awake seizures'' in which patients suffer severe spasms and uncontrollable muscle contractions while maintaining clear mentation. The reason for this was relative sparing at the higher central pathways. Due to these rigorous contractions the sequelae like skeletal muscle rigidity, tachycardia, hyperthermia, respiratory failure, and potentially death through respiratory muscle paralysis occurs. Other complications which can occur include rhabdomyolysis, mixed metabolic and respiratory acidosis, hyperkalemia, and kidney failure. Strychnine is quickly absorbed from the gastrointestinal tract and symptoms can occur within 20 mins of ingestion.[9,10,11] The compound is mainly metabolized in the liver, with varying degrees of renal excretion depending on its serum levels.[9] So far reported median lethal dose of strychnine is approximately 1.5 mg/kg or 50 to 100 mg; Nevertheless, there have been case reports who survived with a serum level of 4700 ng/mL, which is >10 times the serum level seen in some case reports.[11,12,13]

The cause of death in most of the strychnine poisoning cases is due to asphyxia caused by prolonged contractions of respiratory muscles which is a sequalae of paralysis of respiratory centre in medulla.[14] The other proximate causes being cardiac arrest, multiple organ failure, or brain damage.[15]

Symptamatology and Health Effects[15, 16]

Strychnine poisoning has varied presentation from nausea to death depending on the dose/ amount of ingestion. It can take longer time to manifest at about 15 minutes by ingestion compared to inhalation or injection which can manifest within 5 min or less. Respiratory failure and brain death can occur within 15–30 mins with a very high dose and other signs and symptoms develop by ingesting lower doses.

System wise Health effects were tabulated below

S. No	System	Features
1	Central nervous system	Restlessness, apprehension, cold perspiration, heightened acuity of perception, hypervigilance, Tremor, violent repeated convulsions with opisthotonus, impairment of short and medium-term memory
2	Eyes	Exophthalmus, mydriasis, bilateral horizontal pendular nystagmus
3	Skin	Allergic response / Hypersensitivity
4	Respiratory tract	Spasmodic diaphragm movements, cyanosis, dyspnea, hypoxia, respiratory failure
5	Cardiovascular	Weak pulse, tachycardia, hypertension, cardiac arrest
6	Gastrointestinal tract	Vomiting
7	Renal and urinary tract	Myoglobinuria, acute renal failure
8	Musculoskeletal and smooth muscles	Stiffness of facial and neck muscles, hyper-reflexia Contractions of all voluntary muscles simultaneously, including chest and abdominal muscles, hypertonicity of the muscles, tonic twitching of the face and neck muscles, trismus, risus sardonicus, rhabdomyolysis
9	Metabolic	Hyperthermia
10	Laboratory findings	Lactic acidosis, hyperkalemia, elevations of AST, LDH, CPK, leukocytosis

Review of Literature

Literature on strychnine has been reviewed over a period of 100 years (1920-2020) which has been tabulated below.

Refer-ence	Year	Place	Age	No. of cases	Sex	Mode	Available form	Presentation	Drugs	Complications	Outcome
17	1928-32	Indiana, US	Apr-56	11	5 F 6 M	Suicidal (10) Accidental (1)	Tablets	Twitchings, convulsions	Sodiumamytal	Nil	Alive
18	1955	Newyork, US	15 Months	1	Male	Accidental	Laxativepills	Jerks, cyanosis convulsions	Sodium-benzoate	Respiratory failure	Expired
19	1963	Scotland, UK	18 Months	1	Male	Accidental	Tablets	convulsions	Sodiumthiopentone	Respiratory failure	Alive
20	1969	Portugal	1 year	1	Male	Accidental	Tablets	Convulsions, Opisthotonus	Notmentioned (NM)	Respiratory failure	Expired
21	1971	Portugal	13 Months	1	Male	Accidental	Syrup	Rigidity, convulsions	Diazepam	Respir atory failure	Expired
22	1971	Portugal	50 years	1	Male	Suici dal	Tablets	convulsi ons	Diazepam	Nil	Alive
23	1982	Dublin, ireland	Teens	8	NM	Accidental	Whitepowder, cocaine	Pain, stiffness, convulsions	Diazepam	Respiratory failure	7 Alive 1 Expired
24	1984	Portland	49 years	1	Male	Suicidal	Tablets	Unconsciousness, seizures	Diazepam	Respir atory failure	Expired
16	1985	Pittsburg, USA	56 years	1	Male	Suicidal	Rodenticide	Convulsions, bodyrigidity, opisthot onus	NM	Respiratory failure	Expired
25	1986	Portugal	42 yrs	1	Male	Suicidal	Notknown	Cramps, convulsions	Sedation, musclerel axation	Respiratory failure, AKI	Alive
26	1989	Newcastle, UK	32 years	1	Male	Accidental	Fumes, Fire	Convulsions, vomiting	Diazepam	Respiratory failure, AKI	Expired
16	1992	Toledo, USA	14 years	1	Female	Accidental	Syrup	Convulsions, Myalgia	Diazepam	AKI	Alive
27	1992	Malaysia	Teen	1	Male	Suicidal	Pills	Risussardonicus, opisthot onus	Notmentioned	Respiratory failure	Expired
28	1998	Spain	18 years	1	Female	Accidental	Ratbiscuits	Convulsions	Benzodiazepines	Chemicalpancr eatitis	Expired
16	2001	Manitoba, canada	50 years	1	Female	Treatment	Skinlotion	Musclespasms, cramps	Fluids, NAC	Nil	Alive
29	2002	Londoe, UK	42 years	1	Male	Suicidal	White powder	Muscle spasms	Midazolam Pancuronium	Respir atory failure	Alive
16	2003	Negev, Israel	6 years	1	Male	Accidental	Tablets	Cramps, disorientation	Diazepam, Vecuronium	Rhabdomyolysis	Alive
30	2004	Tehran, Iran	28 years	1	Male	Suicidal	Powder	Seizures	Midazolam, Sodiumthiopento ne	Respiratory failure, AKI	Alive
31	2004	Newyork, USA	46 years	1	NM	Suicidal	NM	Musclecramps, convulsions	Diazepam, midazolam, Pancuronium	Respiratory failure, AKI	Alive

32	2010	York, USA	49 years	1	Female	Suicidal	Syrup	Musclespasms	Lorazepam	AKI	Alive
33	2013	Portugal	87 years	1	Male	Suicidal	Powerform	Jerks, Convulsions	Diazepam, sodiumvalproate	Nil	Alive
34	2015	Dutch	47 years	1	Male	Suicidal	Powder	Seizures	Midazolam, Propofol	AKI	Alive
35	2016	Karnataka, India	23 years	1	Male	Suicidal	Seeds	Stiffness, Hyperreflexia	Diazepam	Nil	Alive
36	2016	Atlanta, US	40 years	1	Female	Accidental	Slangnut	Jawpain, Spasms	Barbiturates, Benzodia zepines	Nil	Alive
37	2016	Karnataka, India	36 years	1	Female	Tablets	Treebracts	Tonicclonicseizures, Teethclenching	Anti-convulsants	AKI	Alive
38	2017	Vellore, India	22-39 years	3	Males	Suicidal	Leaves	Vomitings, awake seizures	Lorazepam	Nil	Alive
39	2018	Tehran, Iran	22 years	1	Male	Suicidal	Pesticides	Serialseizures	Benzodiazepines	Respiratory failure	Alive

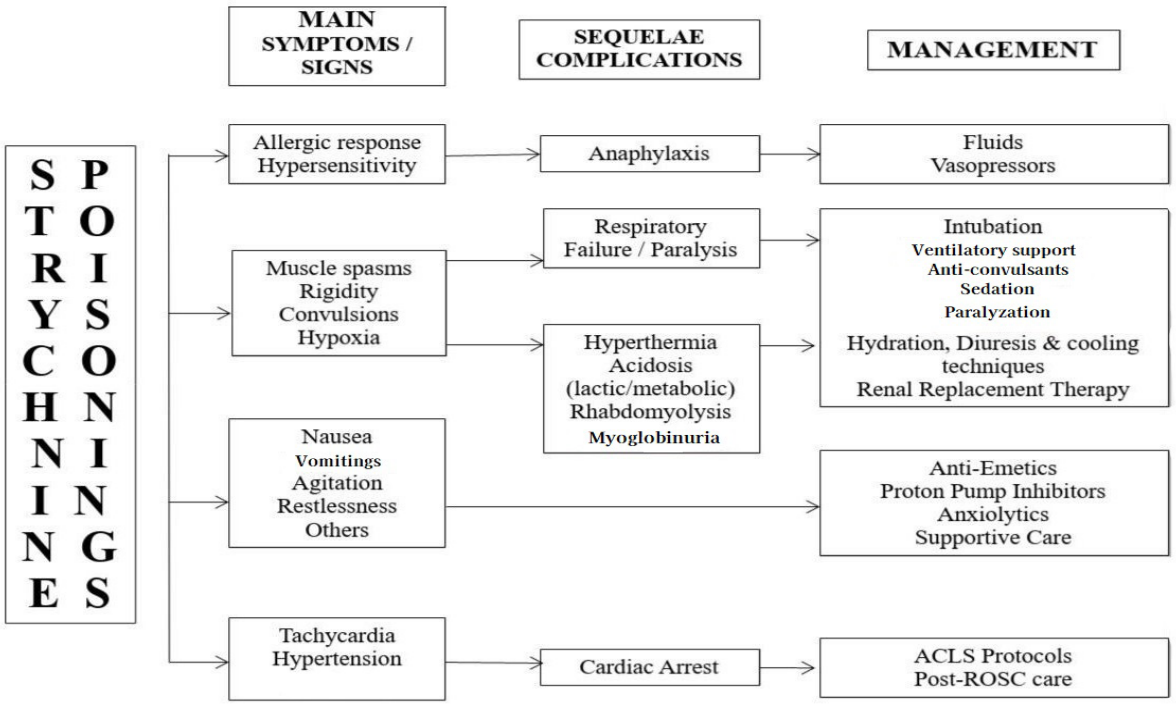
DIFFERENTIAL DIAGNOSIS

The diagnoses which need to be kept in mind includes epilepsy, tetanus, meningitis, rabies, phenothiazine overdose, cocaine and phencyclidine use and exposure to chlorinated hydrocarbons, isoniazid, cyanides, organophosphates or other substances that may cause myoclonus or seizures.[40, 41]

Treatment and its Progrssion From Decades

Observations through the above review of literature: Initially in the age olden days anticonvulsants used was sodium amytal, sodium benzoate and sodium thiopentone. Through the years it has changed to diazepam, lorazepam, midazolam and paralyzing agents like vecuronium, pancuronium etc., Early intubation was adopted now as a good outcome measure. Rapid cooling techniques for hyperthermia were also added some value for recovery of most of the cases. Improvement in renal replacement therapies has lead to avoid the AKI complications. ALL these improvements in care has shown improvement in mortality and morbidity of this toxic ingestion.

(**Antidote** for strychnine poisoning is cipher.) Strychnine poisoning is one entity which is deadliest without a specific antidote but can be treated with proper supportive care. Gastric decontamination can be done if presented early within 2 hrs though there are some evidences stating that it can lead to asphyxia, seizures. Early hospitalization with supportive care including hydration with fluids, medications for convulsions and spasms, cooling techniques and dantrolene sodium for hyperthermia, avoidance of complications like airway compromise, rhabdomyolysis, acidosis, AKI etc., are the mainstay of treatment which can save most of the poisonings of strychnine intoxication as in our case series described below. Regarding medications for convulsions barbiturates and benzodiazepines should be tried. If uncontrolled, sedation and paralysation with early intubation should not be delayed. Proper hydration can avoid rhabdomyolysis and impending AKI. Hyperthermia should be treated aggressively by ice water immersion, cooling blanket or cool mist. Symptoms, complications and management to avoid those were depicted in the below diagram.[16,42]



1: Strychnine Poisoning

Pearls to remember:

- Consider strychnine when history of illicit drug abuse is noted.
- Consider in any case with awake seizures, seizures not subsiding with barbiturates and benzodiazepines.
- Symptomatic treatment and avoiding complications with critical care during first 24–72 hrs can recover most of the cases of strychnine.

CASE SUMMARY AND DETAILS

This is a case series of four members who have consumed strychnine accidentally. Four members (One elderly male, two of them were husband and wife and one elderly lady who will be reffered from hereafter as case 1, 2, 3 and 4 respectively) have gathered at one fine night for a supper arranged at home of case 4 which has become the memorable event for their life time.

She had served the other three and had food later after 20 mins. after a while, case 1 started feeling agitated, nauseating, severe muscle spasms. Within a span of 10-15 minutes he started seizing. Case 2 and 3 were also feeling nausea and not normal. They were being taken to the hospital in the ambulance. Meanwhile case 2 started seizing in the ambulance itself. (Inj. lorazepam has been given in the ambulance for both of them). Just after reaching the hospital case 3 started seizing. We started preliminary treatment for all three cases, while one of us went to elderly lady to take history. The astounding feature about their presentation was awake seizures (conscious and aware of the things happening in the surrounding's during event). Most of the anticonvulsants were tried but seizures dint subside (we strived hard with most of the anticonvulsants but the results were lousy). To avoid complications we have no choice left other than to sedate, paralyze and intubate them. On the other side case 4 was about to finish the history and events happened. Suddenly to our surprise and giving hint, she also had seizures but got subsided with lorazepam. All of them were admitted in intensive care unit (ICU) and treated as per protocols. Treatment included mechanical ventilator (MV) support, anticonvulsants and other supportive care History and treatment part has been tabulated below.

Initial Resuscitation		
Components	**Features**	**Intervention**
A (Airway)	Frothing	Airway protection
B (Breathing)	30/min, decreased saturations, clear chest	ET Intubation, MV support, oxygen.
C (Circulation)	Tachycardia, MAP 65 to 90 mm of Hg	Nil required
D (Disability)	Not assessed	Antiepileptics, barbiturates, benzodiazepines, Sedatives, paralytics infusion.
E (Exposure and Environmental)	Hyperthermia	Cooling techniques
Sample History		
S (Signs & Symptoms)	Nausea, tachycardia, hyperthermia, agitation, restlessness, awareness of symptoms, breathing difficulty	
A (Allergy)	Nil Significant	
M (Medications)	Antihypertensives, antidiabetic medications	
P (Past History)	Nil Such events prior	
L (Last Meal)	Had food half an hour to fourty five minutes back	
E (Events)	Nil Significant	

2: Assessment

Three cases except the case 1 got recovered within a span of 3 days and got discharged within a week. All the details regarding the four cases were tabulated. Case 1 took more time as he had acute kidney injury (AKI) due to rhabdomyolysis caused by seizures.

S. No	Feature	Case 1	Case 2	Case 3	Case 4
1	Onset of symptoms	First	Second	Third	Last
2	Vitals	Stable	Stable	Stable	Stable
3	Drugs used	Benzodiazepines, Sedatives, paralytics	Benzodiazepines, Sedatives, paralytics	Benzodiazepines, Sedatives, paralytics	Benzodiazepines
4	Organ supports	MV support /Renal support	MV support	MV support	MV support
5	MV duration	7 days	3 days	3 days	0 days
6	Complications	AKI / Respiratory failure	Respiratory failure	Respiratory failure	None
7	Cause of complication	Rhabdomyolysis / more muscle mass / more food intake	Seizures	Seizures	None
8	ICU LOS	10	6	6	4

3. Case Wise Involvment

DISCUSSION

By the history at initial presentation provisional diagnosis was kept as Struchnine toxicity, Food poisoning, drug abuse mainly. Strychnine was kept by awake seizures, food poisoning as they had this feature in common before symptom onset and drug abuse as they all gathered for a party. After the recovery of the patients we were able to find the cause by taking detailed history including the examination of food materials used. The cause was found out to be due to seeds used to make dal which are surprisingly strychnine seeds (shown below in fig. 1) which were grown in their back garden. We examined the seeds and confirmed by the forensic team. Later all the events were correlated like symptom onset, complications, recovery with time of consumption of food, amount of consumption respectively.

Fig. 1:

Fig. 2:

In the present case series of four members, they have consumed accidentally Nux vomica seeds which were mix with other seeds from their garden. All the typical features were seen including nausea, awake seizures, hyperthermia, tachycardia etc., Nevertheless, there is no antidote, adequate supportive care along with avoiding complications can show good prognosis as in our case series. Death occurs mainly due to respiratory paralysis which was taken care by early intubation and MV support in our cases.

Point which has to be kept in mind and get cognizance about awake seizures so that mortality and morbidity can be reduced or avoided though strychnine poisoning due to drugs, nux vomica plant products consumption was few and far between.

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