

Strychnine poisoning: a case report

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Abstract

A teenage male was admitted to a hospital in the United Kingdom following the ingestion of strychnine. The typical spasms of strychnine poisoning were observed and he died during the fourth convulsion. The post mortem findings are presented, and strychnine poisoning is discussed.

Key words: Strychnine, poisoning, convulsions.

CASE REPORT

DAS was admitted to the Accident and Emergency department of a hospital at about 6 pm following the ingestion of strychnine half an hour earlier. He had poured the contents of a bottle of strychnine - of unknown quantity, into a mug containing orange squash in the presence of his girl friend. He had previously consumed whisky with his mid-day rice meal.

He was conscious whilst being transported from his home to hospital by ambulance, and had walked unaided from his home to the ambulance. He was said to be "twitchy" but had not developed any convulsions. At initial examination at the hospital he was conscious and was able to give a clear history of the incident. His respiratory rate was normal but with a generalised increase in muscle tone and the reflexes were brisk. During the examination, he developed the first of four convulsions.

In this convulsion, it was noted that there was a generalised increase in the tone in all four limbs with typical opisthotonus and risus sardonicus. The spasm lasted about half a minute and was followed by total relaxation when his muscle tone returned to normal. His respirations also returned to normal.

A few minutes later he developed his second convulsive attack which was longer and more severe. He cried for help. Respiration ceased during the attack but returned to normal after the convulsion. The third convulsion followed shortly afterwards, and was even more severe than the second and was followed by the fourth convulsion during which cardiac arrest occurred. He died 1 1/2 hours later. The ECG, until the arrest, was found to be in sinus rhythm.

AUTOPSY FINDINGS

The autopsy was performed four days after death, the body being preserved at 4°C.

External examination

The body was that of a coloured male 172.5 cm in height with an estimated weight of 70 - 72 kg.

The eyes were natural. There were no subconjunctival petechiae. The ears and nose were healthy as was the mouth. There was no evidence of injury to tongue or lips.

Internal examination

There were a few scattered petechial haemorrhages in the scalp.

Musculo-skeletal system

There was no obvious pathology.

Central nervous system

The vessels at the base and surface of the brain were healthy. The brain and meninges showed severe congestion.

Cardiovascular system

The heart weighed 285 grams. The pericardial sac contained about 200 ml. of pericardial fluid which was blood stained - due to therapeutic procedures. The myocardium macroscopically showed no evidence of fibrosis or infarction. There was a therapeutic puncture mark on the right ventricle anteriorly.

The mitral and aortic valves were healthy as were the coronary vessels, thoracic and abdominal portions of the aorta.

Respiratory system

The larynx and vocal cords showed no pathology. The trachea and bronchi contained blood-stained, frothy fluid material. The lungs were congested and moderately oedematous. There were multiple sub-pleural petechiae in the interlobar fissures on both sides.

Gastrointestinal system

The stomach contained about 50-75 ml of fluid material in which an occasional rice particle could be identified. No powdery material was seen. The gastric mucosa appeared congested but no significance could be attributed to this observation due to the long interval between death and the autopsy. The intestines were healthy. The liver was congested and showed no macroscopic evidence of necrosis. The gall bladder contained bile and the pancreas showed no pathology.

Genito-urinary system

Both the kidneys were congested and healthy. The bladder contained a moderate quantity of urine. The prostate was healthy.

Endocrine and reticulo-endothelial systems

The thyroid and adrenals were healthy. The spleen was firm and congested.

MICROSCOPIC EXAMINATION

Sections of the brain, heart, liver, kidneys, adrenals and spleen showed no congestion. The lungs showed alveolar congestion and oedema.

Toxicology

Specimens of brain, liver, stomach contents, skin fat, kidney, blood and urine were submitted for analysis. However, the analysis was only carried out on the blood, urine and stomach contents. The report was as follows:

Blood alcohol : 48 mg%
 Urine alcohol : 45 mg%
 Strychnine : Stomach contents 530 ug/ml.
 Peripheral blood - 2.4 ug/ml
 Urine - positive

The cause of death was strychnine poisoning. At the inquest H.M. Coroner recorded a verdict of "Suicide".

DISCUSSION

Source

Strychnine is the principal alkaloid of certain members of the Strychnos family of plants and is normally obtained from the dried ripe weeds of *Strychnos nux vomica* which grows in East Indies, India and South-East Asia. It has been introduced to China and Australia and now occurs in scattered areas throughout the tropics and sub-tropics. There are between 150-200 species of *Strychnos*, mostly native to the tropics.

All parts are toxic, containing several alkaloids, primarily and in varying degrees, either strychnine or brucine or both. Strychnine is one of the most powerful poisons acting on the central nervous system. Brucine is similar but less active.'

The seeds are discoid with a central depression, approximately an inch in diameter and about a quarter of an inch thick at the rim. They are grey or green-grey in colour and have satin-like surfaces, because they bear innumerable fine, silky hairs.² They contain about 1.3 - 1.5% strychnine and up to 1.5% brucine. If swallowed whole, a seed will pass through the body without harm. The bark contains no strychnine but 1.68% (when old) and 3.1% (when young) of brucine. Roots possess 0.71% strychnine and 0.28% brucine. The fruit pulp has a very low strychnine content.'

The pharmacopoeial preparations of *nux vomica* and strychnine are *nux vomica* itself; *nux vomica* powder (1.2% of strychnine); liquid extract (1.5% of strychnine); a dry extract (5% of strychnine) and the tincture (0.125% of strychnine). The only B.P. preparations of the alkaloid is liquor *strychnos hydrochlor* (1% strychnine hydrochloride) and syrup ferriphos cum quinine et strychnine - Easton's syrup (0.025% strychnine). The official dose of strychnine hydrochloride is 1/30 - 1/8 gr.³

The availability of strychnine to the general public was severely restricted in the U.K. by drug legislation, in particular the Thirteenth Schedule of the Pharmacy and Poisons Acts 1933 - 64. The sale of strychnine in the U.K. is now controlled by the Poisons Rules 1978 (Schedule 1) and the Medicines (Prescriptions only) Order 1971 - Schedule 1.

Biological effects and mode of action. Absorption and distribution

Strychnine is readily absorbed from the gastrointestinal tract, perhaps more readily from the small intestine than the stomach². It can be introduced by injection or parenterally. Copeman was of the opinion that absorption from the gastrointestinal tract was not rapid and

TABLE 1: Strychnine content in post-mortem tissues (ug/g)

Tissue	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9
Blood	-	0.6	-	nd	61	50	16	+ve	-
Brain	4.2	+ve	-	0.47		5	-	-	-
Kidney	-	0.4	-	0.07	70	90	5	-	48
Liver	108	0.27	-	nd	175	160	209	1.7	110
Lung	-			nd			10	-	-
Spinal Cord	0.1	1.9	+ve	1.8	-	-	-	-	-
Urine	-	-	+ve	7.7	2.5	1	+ve	+ve	-
Stomach contents*	40	108	+ve	1	+ve	100	33	+ve	480

* Total Content

nd -not detected

Kind permission of Oliver JS, Smith H, Watson AA⁵

considerable amounts of the ingested alkaloid will be found in the stomach.⁴ It may become distributed through the body, and fatal effects usually occur sufficiently rapidly to obviate the possibility of the distribution being uniform. Table 1 shows the concentration of strychnine in different tissues.

Elimination

Some of the strychnine is excreted unchanged in the urine and, if the patient survives, excretion may continue for 2-3 days. The greater part is destroyed in the body, principally by the liver.[?] Elimination to some extent occurs through perspiration and saliva.⁴

Strychnine is one of the best known of the agents that increases the excitability of neurons. It causes this effect not by reducing the threshold for excitation of neurons, but by inhibitory transmitters of the neurons, especially the inhibitory effect of glycine in the spinal cord.⁶

It has been described as the most bitter substance known and its taste is detectable in a dilution as high as 1/700,000. More recent reports have, however, assessed 1/400,000 or 1/100,000

as the maximum dilution for taste detection.'

Within minutes the victims will suffer stiffness in the muscles, especially those at the back of the neck, although those of the limbs are also affected. The jaw muscles are affected later. Tremor and twitching then occur rapidly followed by generalised convulsions. Normally, there are five or more convulsions prior to death. Rarely, there may be only one major convulsion prior to death. In the case under discussion the victim arrested and died during the fourth convulsive episode. There is hyperextension of the spine (opisthotonus). The lower limbs are extended and abducted (The lower limbs were slightly flexed in the case of DAS). The arms may be flexed across the chest and the fists closed. A period of complete relaxation follows each fit. Profuse sweating also occurs and the victim complains of thirst. Vomiting rarely occurs. Consciousness is maintained throughout.?

Differential diagnosis

The principle condition that will have to be excluded is tetanus (Table 2). Other conditions may

TABLE 2: Main differences between strychnine poisoning and tetanus

Strychnine	Tetanus
Sudden onset in previous good health - No trauma	Gradual onset with some premonitory symptoms of illness and following recognised trauma
Does not commence in, nor especially affect the jaws	Usually commences in, and especially affects the lower jaw
Relaxation in between fits is quite complete	Relaxation in between spasms never quite complete. Some residual stiffness invariable
Steadily worse/steadily better	Progress rarely steady. Variation and longer remission not uncommon

have to be considered, viz, epilepsy, hysteria and hydrophobia.

Fatal dose

Oral administration of half a grain (32 mg) is generally believed to be the minimal lethal dose for an adult.² Copeman⁴ considered the average fatal dose for an adult to be in the range of 0.5 - 1.75 grains, although smaller doses of strychnine have caused death.

Fatal period

Death within half an hour is unusual and indicative of massive dosage or rapid absorption. On the other hand it is rare for the fatal illness to last as long as twelve hours. Death usually occurs within two hours. If there is survival for three hours, recovery is a probability.'

Treatment

The principles of treatment are to:

- (1) control the fits and relieve the asphyxia,
- (2) remove the poison⁷.

Statham⁸ reported a case of successfully treating accidental strychnine poisoning in a boy aged 14 years. In the management of strychnine poisoning, Statham drew attention to the reappearance of convulsions some 48 hours after initial treatment. He attributed this to strychnine release into the blood stream from the liver.

Pathophysiology of cause of death

Most victims of strychnine poisoning die of asphyxia in the course of a violent convulsion. This is in part a mechanical effect due to prolonged contraction of the muscles of respiration, but it is also due to paralysis of the respiratory centre in the medulla by prolonged stimulation by the poison.' A few, it is stated, die during the period of relaxation at a time when they are severely depressed but, more especially, exhausted.²

Comment

In this case the victim stopped breathing during his second and third convulsive attacks but arrested only on the fourth occasion when he succumbed. Thus it would be reasonable to postulate that the convulsive episodes result in cerebral anoxia which precipitates cardiac arrest resulting in death.

Post-mortem findings

On most, if not all, occasions the examination is entirely devoid of specific findings² Rigor may be detected immediately after death with engorgement of the lungs, brain and spinal cord.⁴ Lloyd and Pedley⁹ are also of the opinion that the autopsy findings are largely negative apart from rather meagre evidence of asphyxia.

The autopsy findings in this case was, in the main, non-specific and in conformity with the reported cases.

Accident-suicide-homicide

The incidence of deaths due to strychnine tends to vary. Oliver et al⁵ estimated that in spite of the introduction of the Pharmacy and Poisons Acts 1933-64 there has been an average of 1 fatality per year in the U.K. over the past 30 years. Bogan et al stated that there have been 92 deaths in England and Wales in the period 1948-61.¹⁰

Most of the deaths appear to be accidental in nature. The most common are the result of the ingestion by children of tablets intended for their parents. Statham⁸ and Hawkins⁷ reported non fatal cases of poisoning, the result of ingestion of strychnine. Haslam reports a non fatal case of poisoning, the result of ingestion of some seven fluid oz (198 ml) of "tonic."

Suicidal poisoning is commonly seen in those with access to strychnine in the course of their occupations - a pharmacist or an agricultural worker. He/she usually takes a large quantity.'

In spite of the bitter taste, even in very high dilutions, cases of homicide have been reported, none though in recent times.² A case of homicidal poisoning by strychnine has been reported in 1966.¹⁰

The toxicity of strychnine is well known and its supply to the public is controlled by legislation. Those engaged in agriculture do however seem to have ready access to it. Thus it is likely that the pathologist and, for that matter, the clinician must always consider strychnine poisoning in those presenting clinically with twitchings, spasms and convulsions or when autopsy fails to reveal a definitive cause of death.

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