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Poisonous Plant (*Gloriosa superb L.*): Its Pharmacological and Therapeutic Profile

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ABSTRACT

Today most of the world population is moving towards herbal medicines. Traditional plant medicines might offer a natural treatment to treat various human ailments. Research work goes to be focused on finding successful results on the therapeutic values of medicinal plants and also exact molecular mechanism of their action at molecular levels. The article gives an overview of Therapeutic and current status of the pharmacological perspectives of *Gloriosa superba L.* a member of lililaceae family, with phytochemicals present in this species were found to have analgesic, anti-inflammatory, anti-thrombotic, anti-coagulant, anti-tumor, "enzyme inhibitor, and anti-venom characteristics". Further clinical studies are necessary to increase our understanding of the links between the documented traditional uses and toxicity of *G. superba*, this article is aimed at compiling an up-to-date medicinal uses and poisonous properties of *G. superba* over its distributional.

Introduction

Botanical Description and Ethnopharmacology

Gloriosa superba L. (family colchicaceae) is not only a notorious human and livestock poison, *G. superba* cause illness and even fatalities to human and animals due to both deliberate and unintentional poisoning. It is local to tropical Africa and south-eastern asia[1]. It is ordinary in forest-savana boundaries, locally common in thickets, hedges, open forest, grassland and bush land, where it can be seen scrambling through other shrubs[2].

Botanical name - *Gloriosa superba* L.

General name and synonyms - Flame lily, glory lily.

Taxonomy of Plant

G. superba is a semi-woody herbaceous, 1 to 4 stems arise from a single V-shaped fleshy cylindrical tuber. The leaves are without stalk, lance-shaped, alternate or opposite or in whorls of upto 3; leaf size: 5 to 15 cm long by 4 to 5 cm wide with parallel veins and tips ending in spiral tendrils which are used for climbing. Large, showy, long-stalked flowers are ready up to of 6 long refluxed petals commonly with wavy margins. Flower size: 6 to 10 cm long by 1 to 2.5 cm wide. Flower colour: generally very bright ranging from red with yellow margins to very pale yellow forms with a mauve or purple stripe; pale white forms also present. Many added colour forms have arisen throughout cultivation. The fruit is oblong, 6 to 12 cm by 2 to 2.5 cm and contains about 20 globose red seeds in each valve[3, 4, 5].

Gloriosa superba

Habitat

The plant grows in sunny positions in free-draining soil; it is very linient of nutrient-poor soils[7] with upto a altitude of 2500 metres above sea level[6]. It is widely grown as an ornamental in cool temperature countries under glass or in conservatories[4].

A native of tropical Africa and is now found growing naturally throughout much of tropical Asia including: India, Sri Lanka, Burma[8]; *G. superba* is also planted outdoors in the southern United States. In cool temperature countries it is treated as a greenhouse or conservatory plant.

Cultivation

The plant can be propagated sexually by seed or vegetatively by dividing the rhizome. Problems during cultivation comprise insufficient pollination, fungal infection such as leaf blight and tuber rot, and crop pestes such as the moths' *Polytela gloriosa* and *Chysodexis chalcities*[9]. Each one split tuber produces only one extra plant in a year's time. *In vitro* experiments with plant tissue culture have been performed[10].

Toxic parts of the plant

The entire plant, mainly the tubers are extremely poisonous. The toxic properties of the plant are essentially due to the highly presence of colchicine[11].

Physico-chemical characteristics

Colchicine occurs as pale yellow to greenish yellow, odourless crystals or amorphous scales or powder. It darkens on contact to light. Melting point is 157°C Solubility in water is about 1/20. It is freely soluble in alcohol and chloroform[12].

Chemical constituent of the plant

In addition to colchicine and gloriosine, *G. superba* also contains other compounds such as 3-desmethyl colchicine, beta-lumicolchicine, N-Formyldesacetyl-colchicine, 2-desmethyl colchicine, chelidonic acid and salicylic acid[13].

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USES

Human Uses

The alkaloid rich plant has been as a conventional medicine in many cultures. It has been used in the treatment of gout, infertility, open wound, ulcer, arthritis, cholera, kidney infection, typus[6], itching, leprosy[14], bruises, sprains, haemorrhoids, cancer, impotence, nocturnal emission[7], smallpox, sexually transmitted disease and many type of internal parasites[7], Such as anthelmintic[15]. It has been used as a laxative and an alexiteric[16]. The juice of the leaves is used to kill head lice and also as an ingredient in arrow poisons [17]. In pregnant women, it may cause abortion. In part of India, extract are applied topically during child birth to reduce labour pain[6]. The flower is part of religious rituals. It is also the state flower of Tamil nadu[18]. Miscellaneous pharmaceutical product and other therapeutic preparation.

Description

Different parts of the plant have a broad variety of uses especially within traditional medicine practised in tropical Africa and Asia. The plant is sometimes used as an adulterant of aconite (Aconitum sp.). The tuber has normally been used as a suicidal agent among women in rural region and it has also been used for homicide.

The tuber also claims antidotal properties to snake-bite and in India it is generally placed on windowsills to deter snakes. Many cultures accept as true the species to have various magical properties.[19, 4, 5].

Circumstances of Poisoning

High risk circumstances

In region of tropical Africa and Asia the tubers of *G. superba* may be wrongly eaten in place of Sweet Potatoes (*Ipomoea batatas*) since the former is a weed of farmland and the tubers look like those of Sweet Potatoes.

High risk geographical areas

The highest risk areas are likely to be throughout the natural variety of the species (i.e. tropical Africa and Asia, including Sri Lanka). Accidental experience to the plant may also occur in cool temperate countries of the West where it is grown as an ornamental.

Summary

Review of clinical effects

Initial symptoms develop within two to six hours after taken of tubers part of *G.superba*. They are characterized by numbness and tingling in the area of the mouth, burning and soreness of the throat, nausea, intense vomiting, abdominal pain and bloody diarrhoea leading to dehydration. The other important difficulties that follow may consist of: respiratory depression, dyspnoea, shock, hypotension, marked leucopenia, thrombocytopenia, coagulation disorders, haematuria, confusion, seizures, coma and ascending polyneuropathy. Alopecia and dermatitis are the late manifestation that develops about one to two weeks after poisoning.

Diagnosis

Bio-medical analysis: daily full blood counts, coagulation tests, serum electrolyte levels and urinalysis are the important investigations to measure the clinical condition. Blood collection for colchicine dosage has to be kept in the dark with anticoagulant.

First-aid action and management principles

If the patient is conscious and alert, induce vomiting by tickling the back of the throat or by giving syrup of ipecac:6 to 18 months - 10 mL, 18 months to 12 years - 15 mL) followed by 1 to 2 glasses of water to induce vomiting. Repeat after 15 minutes if no response. If ipecac is not available or if the patient has not responded in 5 minute after the second dose or in an adult, take out a stomach wash out.

The patient should be admitted to a hospital immediately with, if available, vomit and any remaining plant material.

Management principal

Carefully monitor the respiration. Ensure adequate airway. Perform gastric lavage immediately. Anticipate and treat hypotension with sufficient intravenous fluids and vasopressors. Blood transfusion will also be useful to support the circulation. Continuous cardiac monitoring is useful. Correct dehydration and electrolyte imbalance. Monitor renal function. Initial forced dieresis enhances elimination of colchicine and should be performed once dehydration and shock is corrected. Keep the patient under observation.

Routes of Exposure

Oral

Kinetics

Absorption by route of exposure colchicine is readily absorbed from the gastrointestinal tract. Absorption may be modified by pH, contents in the stomach and intestinal motility.

Distribution

Colchicine is actively taken up intracellularly. Approximately 50% circulating colchicine is bound to plasma proteins. The apparent volume of distribution exceeds total body water (2.2 ± 0.8 1/kg)[20].

Biological half-life

Colchicine has an extremely short plasma half life of about 20 minutes[20].

Metabolism

Colchicine is partially deacetylated in the liver although as much as 20% may be excreted unchanged by the kidney. Bulky amounts of both colchicine and its metabolites are subjected to enterohepatic circulation[20].

Elimination and excretion

Colchicine and its metabolites are excreted in urine and faeces[21].

Toxicology

The plant has poisonous, toxic enough to cause human and animal fatalities if integrated. It has been used to commit murder, to reach suicide[26], and to kill animals[8] every part of the plant are toxic, especially the tuberous rhizomes. As with other member of colchicaceae, this plant contains high level of colchicine. It also contains the alkaloid gloriocine. Within a few hours of the ingestion of a toxic quantity of plant material, a victim may experience nausea, vomiting, numbness, and tigling around the mouth, burning in the throat, abdominal pain, and bloody diarrhea, which leads to dehydration[13], colchicines is known to cause alopecia.

Mechanism of action

Colchicine affects cell membrane structure indirectly by inhibiting the synthesis of membrane constituents[21]. It binds to tubulin (the structural proteins of microtubules) preventing its polymerization into microtubules. This anti-miotic property disrupt the spindle apparatus that separate chromosomes during metaphase. Cells with high metabolic rates (e.g. intestinal epithelium, hair follicles and bone marrow) are the most involved by the arrest of mitosis. Colchicine also has an inhibitory cause on various phosphatases[21]. Gloriosine also has an anti-mitotic effect[11].

Toxic dose of colchicine

Reported that the lethal dose of colchicine for man may be about 60 mg although smaller amounts have also caused

death[22], has reported a patient who survived after ingestion of 350mg of colchicine tuber[23,11].

Relevant animal data

LD_{50} of colchicine for rats was 5 mg/kg[24].

Clinical Effects

Acute poisoning

The commonest clinical presentation of poisoning is severe gastroenteritis with nausea, vomiting, diarrhoea with blood leading to lack of fluids, hypovolaemic, shock and acute renal failure. Muscle weakness, hypoventilation, ascending polyneuropathy, bone marrow depression and coagulation disorders are the other characteristics of poisoning. Death in severe poisoning occurs due to shock or respiratory failure although haemorrhagic or infective complications may cause death after the first day.

Systematic description of clinical effects

Cardiovascular

Heart - there is no direct effect on the heart, but fluid and electrolyte disbalance, often causes hypovolaemic shock manifested by hypotension and tachycardia.

Respiratory

Respiratory failure is thought to be due to the paralysis of intercostal muscles rather than the
direct depression of the respiratory centre by colchicine[25]. The patient may be dyspnoeic and cyanotic.

Central nervous system (CNS)

There is progressive paralysis of the central nervous system and peripheral nervous system [26]. Confusion and delirium may occur either secondary to poor cerebral perfusion or as a result of direct cerebral toxicity[19]. It may also cause convulsions, restlessness and coma.

Peripheral nervous system

Ascending polyneuropathy, weakness, loss of deep tendon reflexes may be described.

Skeletal and smooth muscle

Colchicine could have a direct toxic effect on skeletal muscles causing muscular weakness. Rhabdomyolysis may occur with major increase in muscle enzymes and myoglobinuria as a result of direct muscular damage. Muscle weakness that may persist for many weeks may contribute to respiratory deficiency[19].

Gastrointestinal

Gastroenteritis including nausea, vomiting and diarrhoea with blood accompanied by colic and tenesmus. Loss of fluids and electrolytes leads to hypovolaemia. Intestinal ileus may develop within the first few several days and may persist up to a week[19].

Renal

Any direct toxic effect of the toxin on kidney is not clear. Renal failure is probably secondary to excess fluid loss or hypovolaemia and is proceed by oliguria and haematuria. Proteinuria could also occur[26].

Endocrine and reproductive systems

Vaginal blood loss has been reported as a feature of intoxication. Tubers are used as an abortifacient in a few countries.

Dermatological

Alopecia usually occurs one or two weeks after the taken of *G. superba*. A case of generalize depilation has also been reported [11]. Desquamative dermatitis has been reported as another dermatologic manifestation[23].

Eye, ear, nose, throat: local effects

Subconjunctival haemorrhages have been observed[11].

Burning and rawness of the throat may be early symptoms of toxicity.

Haematological

Colchicine has a depressant effect on the bone marrow which is characterized by a transient leucocytosis followed by leucopenia. It could also cause thrombocytopenia that may give rise to different coagulation disorders resulting in vaginal bleeding, gastrointestinal haemorrhages. Chronic thrombocytopenia occurring within 6 hours of poisoning has been documented[27]. Anaemia may occur, mostly secondary to haemorrhage.

Immunological

Patients are prone to infections as a result of leucopenia.

Fluid and electrolyte disorder

There is an extensive fluid and electrolyte loss due to extreme vomiting and diarrhoea or sometimes due to haemorrhages. Hypokalaemia, hypocalcaemia, hypophosphataemia and hyponatraemia may present[26].

Management

General ethics

Hospitalize the patient immediately. Constant and extended monitoring monitoring is important. Ensure adequate ventilation. Before instituting symptomatic and supportive therapy remove the plant material from gastrointestinal tract by emesis or gastric lavage without delay to reduce additional absorption. Give adequate intravenous fluids. accurate any electrolyte imbalance. Maintain a fluid balance chart. Specific measures should also be taken for the management of shock. Cardiac monitoring is useful.

Hypotension and shock

Fluid loss may lead to hypovolaemic shock with hypotension: Manage hypotension as required and give appropriate oral fluid. Make sure a clear airway, advance ventilation and give oxygen (Ha 4 + 5 + 6). Sudden haemodynamic monitoring is very helpful[27].

Renal failure

Renal failure with oliguria is a common characteristic. Maintain an adequate urine output with plenty of intravenous fluids. Established renal failure may need peritoneal or haemodialysis.

Leucopenia

Fresh blood transfusions are essential to correct leucopenia. Prophylactic anti-biotic therapy is advisable if leucopenia is present.

Coagulation disorders

If clotting time is abnormal, vitamin K and fresh frozen plasma should be given. Haemorrhagic manifestations should be treated with fresh blood transfusions.

Enhanced elimination

Forced diuresis, if instituted early should be of benefit by eliminating colchicine. Haemodialysis, haemoperfusion and other relevant measures are of no value since of large volume of distribution and limited renal excretion of colchicine[19].

Antidote/antitoxin treatment

There is no specific antidote available, but immunotherapy (fragments fab) is available for clinical trial on humans in some countries(France).

Illustrative Cases

A 21 year old married woman, who was said that she have taken about 124 g of tuber (total amount of colchicine about 350 mg), feeled gastrointestinal symptoms in 2 hours. On admission, about 24 hours after eating, she was unconscious and dehydrated. Her blood pressure was 95/70 mmHg; pulse

rate was 122/minute and the respiratory rate was18/minute. She developed acute renal failure, menorrhagia, and after 11 days, marked generalized alopecia.

She finally recovered and after two months her scalp hair showed regrowth. Pubic and axillary hair also showed regrowth, though the latter remained very scanty.

Reported another non-fatalcase of an 18 year old girl who had eaten uncooked tubers. Six hours after intake she developed harsh gastrointestinal symptoms, vaginal bleeding, acute renal failure, quickl ascending polyneuropathy, respiratory distress, nonappearance of tendon and plantar reflexes, leucopenia, alopecia and dermatitis. She fully improved in four weeks[24].

Conclusion

Traditional healers seem to be aware of its toxicity as the amounts they prescribe are such that toxic symptoms are minimized. Using larger doses usually result in poisoning human. On the basis of current information and evidence, *G. superba* extract are characterized by instances of toxicity and it should be used under supervision of physician.

There is need for further research, clinical trials and product development. However, there is a need to study the acute, sub-acute, chronic toxicity and pharmacological safety associated with the use of *G. superba* as medicine.

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