

Multi Organ Dysfunction following Consumption of tubers of *Gloriosa Superba*: A Rare Case Report

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ABSTRACT

25 years married man was admitted on to emergency acute medical care unit. He had fallen ill after an afternoon meal from 4 days ago which consisted of rice and 3 tubers identified as *Gloriosa superb*. About 3hrs after the ingestion, he developed abdominal pain, vomiting (15-20 episodes), and watery diarrhea 15-20 episodes. He developed bleeding gums, yellowish discoloration, hematemesis 30-50 ml, 24episodes, haematuria, decreased urine output along with continued diarrhea and vomiting. He has treated with iv fluids crystalloids and colloids titrated, 1 fresh frozen plasma (FFP), 1 fresh platelet transfusion, inj. Vit K, supportive treatment with close monitoring of vitals. Therapy as planned for ventilator support, however, had ever had to bleed per rectum and hematemesis with aspiration, even with best available resuscitative efforts he succumbed to his illness and declared clinically dead after 36hrs of admission, his cause of death was given as multiorgan dysfunction see to *Gloriosa superb*. Though poisoning with *Gloriosa superb* is rare it is fatal and requires aggressive and prompt treatment is mandatory from saving the patient from death.

Key words: *Gloriosa superb*, multiorgan dysfunction, bleeding manifestations, fresh frozen plasma, death.

INTRODUCTION

Gloriosa superb is a climbing hedge plant growing all over India mostly in southern part, also known by name glory lily, flame lily, pulasapadu pappu gadda, gharba inatin in Sanskrit meaning which cause abortion. A medicinal plant cultivated for colchicines used for various ailments like Joint pains. Colie, antifertility, abortifacient, leprosy, anthelminthic, gonorrhoea to name to a few.

Gloriosa Flower



Fruits of Gloriosa



Tuber of Gloriosa



Zimbabwe has declared gloriosa as national flower and Tamilnadu as the state flower. Fresh tubers are more poisonous contains 6mg of colchicines per 10grams of the tuber,also have other alkaloids gloriosine and resins.

Primary actions of *Gloriosa* are:

1. Blocks mitosis at metaphase in dividing cells especially GI tract, bone marrow.

2. Reduction of neutrophils migration, phagocytosis, adhesion and chemo taxi

- Negative inotropic effects, decreased calcium myofilaments sensitivity decreased sarcoplasmic reticular on cardiac muscle.
- Neurotoxicity by decreased function axonal transport and vesicle release

Clinical features can be divided into

Stage 1 0-24 hrs	Nausea, vomiting, diarrhea, abdominal pain, anorexia, peripheral leucocytosis, electrolyte imbalance
Stage 2 24hrs-7d	Pancytopenia, coagulopathy, cardiotoxicity, ARDS, sepsis, Rhabdomyolysis, myopathy-proximal muscle weakness, neuropathy-ascending paralysis, bone marrow hypoplasia, mental state changes.
Stage 3 7d	Reversible alopecia, reversible azoospermia, peripheral neuropathy, skin changes

Treatment includes

Gastric lavage with activated charcoal, a large volume of crystalloids and colloids, FFP, platelet, blood transfusions, inotropic support for cardiogenic and circulatory shock, treatment of electrolyte imbalance, GM-CSF for pancytopenia, mechanical ventilation for ADRS with O₂ inhalation, antibiotics for sepsis, dialysis for renal failure. Since alopecia and azoospermia are a reversible wait and watch therapy is used. Reports of colchicines specific fab are published but availability, the success rate is to be measured.

CASE REPORT

25 years married man was admitted on to emergency acute medical care unit of S.V.R.R.G.H, Tirupati, Andra Pradesh. He had fallen ill after an afternoon meal from 4 days ago which consisted of rice and 3 tubers identified as *Gloriosa superba*. The colchicine content of the fresh tubers was 0.3%. Total amount consumed was of 300 gm of the tuber with colchicines 180 mg.

About 3hrs after the ingestion, he developed abdominal pain, vomiting (15-20 episodes), and watery diarrhea 15-20 episodes. He was treated at a local hospital for 4 days and referred to our hospital. He developed bleeding gums, yellowish discoloration, hematemesis 30-50 ml, 24episodes,

haematuria, decreased urine output along with continued diarrhea and vomiting.

On examination he was conscious, restless, dehydrated with toxic look, pulse rate 108, moderate volume in sinus rhythm, B.P 100/70 mm of Hg, R.R of 18/m, general examination showed pallor, icterus, mild pedal edema, bleeding gums, nonpalpable purpura, ecchymotic patches on both upper limbs, subconjunctival hemorrhage. Lungs showed fine inspiratory crepitations, tachycardia. Abdominal examination revealed diffuse tenderness, mild distension, hepatomegaly with respiratory seizures examination, 3cms below ICM, soft tender with sluggish bowel sounds.

Investigations revealed

Hb	8.4 gms
Dc	74N,21L,05E
BT	3.40
Platelets	26000
PT/INR	1.4
Bilirubin	1.0
Direct	0.3
SGOT	235
SGPT	230
ALP	64
Proteins	6.4
Alb	2.7
Urea	32
Creatinine	0.9
Na	124
K	4.8
Cl	99.8
Uric acid	4.7
Using abdomen	Minimal free fluid include sac distended loops s/o partial obstruction
ECG	ST elevation V1-6,T interval 2,3,av F f/o myocarditis
C/X RAY	Prominent broncho vascular marking No pulmonary edema
Urine microscopy	Plenty of RBC/HPF



He was treated with iv fluids crystalloids and colloids titrated, 1 fresh frozen plasma (FFP), 1 fresh platelet transfusion, inj.vit k, supportive treatment with close

monitoring of vitals. next day he developed heavy about of hematemesis 200ml fresh blood, anuria past 12 hours, vitals showed PR 124/min, B.P 80/6 Ommof Hg, tachypnoea, spo2 of 74% with 6lts of O2, was put on inotrope support nor adrenaline and dopamine titrated to his vitals. 1FFP was transfused along with 1packet of blood. Investigations showed following

platelets	20000c/cu mm
bilirubin	2.0mg
indirect	1.2
creatinine	1.4mgs

Therapy as planned for ventilator support, however, had ever had been bleeding per rectum and hematemesis with aspiration, even with best available resuscitative efforts he succumbed to his illness and declared clinically dead after 36 hrs of admission, his cause of death was given as multiorgan dysfunction see to *Gloriosa superb*.

CONCLUSION

Though poisoning with *Gloriosa superb* is rare it is mostly fatal and requires aggressive and prompt treatment is mandatory from saving the patient from death.

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